TRANSACTIONS

OF THE

THIRTY-THIRD ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

HELD AT

ST. LOUIS, MO.,

OCTOBER 15-19, 1928

Kijc
Amer Acad of Ophthalmology and
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8-22-1929

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JOINT SESSION

PRESIDENT'S ADDRESS.

LUTHER C. PETER, M.D.

PHILADELPHIA, PA.

Mr. Chairman and Fellow Members of the Academy: I take this opportunity of expressing to you my deep appreciation of the honor of being called upon to preside over the Academy in the year 1928. It is with no little pride, but with much temerity, that I add my name to the list of names appearing upon the handle of the gavel, which each presiding officer is privileged to hold as a coveted possession for a period of one year.

For those who are not familiar with the history of this gavel, may I remind you that it was made from the operating chair of the first President of the Society, a resident of this City, the distinguished Adolph Alt. It was presented to the Society by our much respected and lamented Lee Francis, who was the presiding officer in the year 1920. Aside, therefore, from the honor of acting as custodian of the gavel for this year, I count myself fortunate in being privileged to use it during the session in this City, because it calls to mind the memory of two of our esteemed colleagues and the rôles which they played in the development of this wonderful organization.

If I fail to live up to the ideals, as embodied in the lives of the distinguished men who have preceded me in this coveted position, or fall short of what you, my fellow confreres, may rightfully expect, it will not be because of a lack of honest effort to give my best to the Society. You will pardon a personal reference, when I recall to you that my introduction into the Academy was thru one whose memory we still cherish because of his contagious enthusiasm, his scholarly attainments, his loyal friendship, and his great interest in the Academy—the late Wendell Reber. If I have been able to contribute, even in a small way, to the Academy's activities, it was due largely to a desire to carry on, as well as lay within my power, the work which he was obliged to relinquish by his untimely death.

In casting about for a suitable subject upon which to address you, it seemed as tho the wealth of the material which appears

upon the program almost forbade the addition of another contribution of a scientific character. Inasmuch, however, as a presidential address should be of this character, it may not be inappropriate to discuss certain relations which nasal and otitic pathology bears to ocular disease.

Altho the specialties represented in this organization are closely related, and deal with diseases of special sense organs which anatomically and physiologically are in a measure akin and in juxtaposition, there are definite lines of cleavage which separate them. In some instances, by choice, each has formed the basis of a distinct and separate specialty; in other instances, by necessity, they are combined as a special group. Aside from the fact that they logically form a convenient group, which, under certain circumstances, is practical as a specialty, there are contacts and relations which will bear stressing for the man who includes all in his practice and for the man who practices one as a specialty. There are other reasons why a discussion of some of these relations is timely. In some medical centers, there is a tendency to minimize this relation, and even to fail to find any cause and effect relation of sinus disease to optic neuritis, papillitis, and edema of the nervehead. Furthermore, when these conditions occur, as they do in the experience of our American confreres, as well as in most European centers, cooperation between ophthalmologist and rhinologist must be prompt, with a careful weighing in the balance of certain facts from the standpoint of each.

Disturbed Ocular Motility.

Disturbance of ocular motility, as the result of nasal disease or of nasal injury, is more common than the literature would indicate. Paralysis of the internal rectus muscle following intraocular operations are recorded by myself (The Extraocular Muscles, Peter, p. 225), by de Schweinitz and others. Greenwood reported an ocular paralysis following the tamponing of the nose. Bordley cites a Weber syndrome following the improper passage of a eustachian catheter. I personally observed one case of paralysis of the internal rectus which disappeared within twenty-four hours after opening of diseased ethmoid cells. The relation of the phorias to disease of the nasal sinuses has received scant attention, altho of frequent occurence. In one of my cases of 18 diopters of exophoria, drainage of the sphenoethmoidal cells brought about complete recovery. Lesser degrees of exophoria have been similarly relieved. One of my most recent cases was

that of a young man who suffered severe ocular pain which was increased on lateral rotation. An acute cold in the nose which occurred one week previously suggested the advisability of a nasal study. The first report read: "Sinuses negative." A second request was made and the report read: "Acute ethmoiditis." The pain and disturbed ocular motility disappeared after suitable nasal drainage. In searching, therefore, for the cause of disturbed ocular motility, a careful investigation of the nasal sinuses should be included in our routine studies.

Uveitis.

A phase of ocular pathology which has been much discussed is that of uveitis and its relation to sinus disease. When, by a process of elimination, uveitis seems to be due to infected tonsils, the ophthalmologist has little trouble in finding some one to remove the offending organs. The cause and effect relation is patent to all and needs no stressing. When, however, by the same process of reasoning, pathology of the sinuses is suspected as a cause, the problem often becomes a serious one, largely because there may be no gross evidence of disease of the sinuses, and the rhinologist hesitates to invade cells which seem to be normal. There is good ground for conservatism in nasal surgery. In this dilemma, however, there are two who are vitally concerned, namely, the patient whose vision is slowly but surely becoming permanently damaged or lost, and the oculist to whom the patient looks for relief. The matter should reduce itself to a frank conference between the ophthalmologist, the rhinologist, and the patient, in which the dangers should be frankly discussed. If, by a process of elimination, the usual probable causes of uveitis can be excluded, especially when the ocular disease is unilateral, it would seem that conservative exploratory surgery will offer less risk to the patient's welfare than permanently damaged vision.

A few concrete cases will best illustrate the matter. I will cite but two which are illustrative of a large group which might be discussed with profit if time permitted. The first is that of a man of about 35 years of age, whose history I reported before the Pennsylvania State Medical Society in 1921. Briefly, the case was one of bilateral uveitis which went on to blindness, the fields showing contraction in the superior temporal quadrants first, not synchronously but consecutively. Two well known rhinologists reported the sinuses as negative and refused to

operate. A third found both sphenoids diseased, operated, and uncovered well advanced bone necrosis. The operations were performed too late to save the sight, altho for two years I was firmly convinced that the uveitis was of sinus origin. During the last three years, I have had under my care a nurse who had an intractable unilateral uveitis. By careful studies, other sources of infection were excluded, and again the sinuses seemed to be the source of the trouble. Repeated examinations by rhinologists failed to uncover any evidence of nasal disease, and after each examination, the rhinologic surgeon advised against operation. About six months ago, I succeeded in having the sphenoids opened. In each sinus there was ample evidence of disease of long standing. Since operation, the patient has made a good recovery from her uveitis. For the sake of brevity, I quote but two cases. They represent many who have come under my observation.

In order to strike a balance sheet of this group, one might place on one side of the line: 1—In a fair number of these cases, there is no definite evidence of disease of the sinuses without surgical exploration. 2—The sinuses should not be subjected to surgical measures without good indications for the same. On the other side of the line may be placed the following items: 1—The patient's vision is gradually diminishing, with prospects of irreparable damage and possibly total loss if the cause can not be removed. 2—By a process of elimination, all probable sources of infection but that of the sinuses can reasonably be excluded. 3—If the uveitis is unilatral, the focus of infection may or may not be close at hand, but the condition presumably points to a near-by infection. When this array of facts can be placed before the patient, there are few who will not take the chance of having their sinuses explored, providing the work is performed by one who is competent in sinus surgery.

Optic Nerve Involvement.

As serious and as difficult as it is to determine the source of infection in some of the groups of cases just discussed, there is another series which demands even closer attention and prompter action. They are the cases in which the optic nerve becomes involved: retrobulbar neuritis, papillitis, neuroretinitis and papilledema. In the former groups, there is ample time for mature deliberation; in the latter, the necessity for prompt and definite action must be appreciated by the surgeon, if the patient

is to be safeguarded against permanent visual loss as the result of postneuritic atrophy. In my experience, unilateral retrobulbar neuritis, neuroretinitis, papillitis, or papilledema, even of moderate degree, calls for prompt action when sinus pathology is the etiologic factor. It is alleged that many of these cases make a spontaneous recovery, with only a moderate degree of residual atrophy and little or a negligible amount of visual field disturbance. I find it difficult to agree that even a moderate degree of residual atrophy is negligible. Careful field studies by precise methods, with colored stimuli and minute white test objects, reveal disturbance of the fields which is of serious import. In fact, the degree of the residual atrophy can clearly be demonstrated to bear a very definite relation to the promptness with which surgical measures are instituted to correct the sinus pathology. Because of the resulting damage to vision, the necessity for prompt and comprehensive studies becomes imperative. A patient so afflicted should be hospitalized immediately, and should be subjected to the complete studies with which we are all familiar. Unfortunately, it is not always possible to determine whether the optic nerve disease is due to an infection in the general circulation, arising from diseased tonsils or remote foci of infection, or whether it is due to sinus involvement. In the interests of the patient, the proper method of procedure is to direct attention to each of the possible factors in the case. Inasmuch as sinus disease is probably the most serious in causing permanent damage to vision, it should be the first to receive most searching study, unless some other focus is manifestly the cause of the trouble. As in the obscure cases of uveitis, simply inspection of the nasal cavities may not reveal either the presence or the extent of the sinus pathology, which may be uncovered by surgical measures. The number of cases which might be cited in support of this statement is large. A few will suffice, and these few may recall to each of you other cases similar in character.

One of unusual interest is that of a man of about thirty-five, who found his sight rapidly growing dim in the right eye. A fundus study revealed a unilateral papillitis with a swelling of about three diopters. Two examinations by a rhinologic surgeon of known ability in sinus disease failed to uncover any nasal pathology. Notwithstanding the negative character of the nasal findings, by a process of elimination, the sinuses were thought to be the source of the trouble. A request was made "to wash the

sphenoid if possible." The rhinologist facetiously remarked to a bystander that he "was operating upon a normal sinus at the request of the ophthalmologist." The so called normal sphenoid proved to be pathologic, and vision rapidly improved twenty-four hours after the operation. The usual residual atrophy followed, which might have been less had the sphenoid been opened two weeks earlier.

Before an audience of this type, it is unnecessary to multiply case histories to establish the fact that disease of the sphenoids and ethmoids is responsible for many cases of retrobulbar neuritis and other forms of optic nerve disease. Of more vital importance is a discussion of the conservation and preservation of vision by prompt cooperation between the rhinologist and ophthalmologist, in instituting early and effective surgical measures to correct or eradicate the nasal pathology. A study of a series of cases will convince the most skeptical and conservative surgeon that the residual atrophy and consequent visual disturbance bears a direct relation to the length of time which is allowed to elapse between the first appearance of the ocular symptoms and the time when corrective nasal surgery is instituted. A recital of a few cases fresh in the mind of the speaker will illustrate the point.

A medical student reported with a unilateral reduction of vision from 6/5 to 6/15 in three days previous to the examination. There was a definite optic neuritis present. Before the Roentgen ray report could be made, the rhinologist decided to infract the turbinate body to establish better drainage. There was an immediate improvement in vision, which, however, again relapsed after several days. The X-ray plates revealed definite pathology of the sphenoidal sinus. Prompt opening of the sphenoid was followed by permanent restoration of vision. There was no residual atrophy. No time was lost in instituting nasal surgery in this case. A day's delay in the first operation might have reduced the surgical measures to a single operation as indicated by the X-ray report and the pathology which was uncovered in the sphenoid.

A second case occurred in a woman of fifty. Vision in one eye was reduced to 6/15, but the patient failed to report for two weeks after its discovery. A third week was lost because of the illness of the rhinologist. There was a marked unilateral neuroretinitis present, which increased during the week of observation. Definite sphenoidal pathology was uncovered by operation, and

vision improved. Subsequent treatment by elimination brought the vision up to 6/6 partly. There was, however, a gradual recession of vision to 6/12, with the appearance of definite optic atrophy. The form field was moderately contracted with considerable loss of the color fields. The patient's ability to read at the near point has fallen to type 2 on the Jaeger card.

A third case in natural sequence is that of a woman of thirty, who developed visual loss in one eye over a period of a week. The ophthalmoscope revealed a swelling of the nervehead of three diopters. There was an absolute central scotoma present, and field changes as shown in Fig. 1. The patient was admitted to the hospital for operation and marked sphenoidal pathology was uncovered. The subsequent progress and recovery of the patient is best shown in Fig. 2. About ten days elapsed between the first appearance of visual disturbance and the time of operation, but nevertheless atrophy appeared, less than in the former case, but definitely present. I take pleasure in recording this case because it is a definite instance of papilledema due to sinus disease—a much disputed complication of sinus disease.

A fourth case will illustrate the outcome in a larger group. A patient of fifty discovered that she suddenly (over night) became blind in one eye. A week elapsed before she consulted a physician. There was a marked neuroretinitis present which rapidly increased during the week of operation before she consented to enter a hospital. There was no light perception present in any part of the field. Two weeks elapsed in this case between the apparent onset of blindness and the date of the nasal operation. The rhinologist found sphenoiditis of long standing with marked bone pathology. After the operation, the neuroretinitis subsided, but only peripheral temporal vision returned. It is probable that there was some disturbance of vision before the sudden onset of blindness, but if so, the patient was unaware of it as often is the case.

The citation of four cases does not warrant the drawing of conclusions as to the relation of the degree of atrophy to the time which elapsed between the onset of ocular symptoms and the time of intranasal surgery or treatment. Many factors should be considered which might modify such conclusions. The four cases are submitted, however, as illustrative of groups of cases in the experience of an individual surgeon, from which certain definite conclusions may be drawn,—conclusions which seem to be borne out by the literature.

The conclusions are: 1—There is a definite cause and effect relation between sphenoethmoidal disease and optic nerve pathology. 2—This cause and effect relation calls for the closest cooperation between the ophthalmologist and rhinologist. 3—The cases which I have been fortunate enough to observe unmistakably indicate, that when optic nerve disease is due to sphenoethmoidal pathology, promptness in instituting measures which will relieve the nasal disease is apt to lead to a minimum amount of residual optic atrophy.

Ocular Pathology in Cavernous Sinus Thrombosis of Nasal and Otitic Origin.

Finally there is a common field in which ophthalmologist and otorhinologist share equally in responsibility and ability in saving life, if the cooperation is close, namely, in cavernous sinus thrombosis of nasal or of otitic origin. There are forms of this fatal condition in which the surgeon can be but an interested but helpless witness of the approach of death. In those cases, however, which have their origin in the nasal sinuses, or in otitic disease, and the approach is thru one of the anterior routes, prompt cooperation between the ophthalmologist and otorhinologist has and will save many lives. The relative frequency of sphenoethmoidal and of otitic origin, with orbital abscess as a complication, may be gleaned from Eagleton's comprehensive studies of twenty-five cases. (Eagleton on "Cavernous Sinus Thrombophlebitis.") In that group, four originated in the nasal sinuses and three were otitic in origin—28 per cent. Within the year, I had two cases in the hospital at the same time. Thru prompt cooperation on the part of the rhinologist, and by combined surgery, both cases made a good recovery. Fortunately, some of these cases begin as a parietal type of thrombosis, the peripheral walls of the sinus becoming involved first with a gradual development of complete thrombosis. If the necrotic focus of infection in the sphenoethmoidal calls is promptly opened and orbital drainage is established at the same time, recovery may follow. In these conditions, there can be no reasonable ground for difference of opinion as to the necessity for closest cooperation and the need for immediate surgical intervention. It is a common belief that cavernous sinus thrombosis terminates fatally and recovery is a rare exception. From a review of the literature and from my personal experience, this conclusion seems to call for considerable modi-

fication. Two types offer a more hopeful outlook; they are, first, those in which the thrombosis has its origin in the blood stream —a systemic condition; and second, those cases in which the infection springs from the sphenoethmoidal cells or from the middle ear. When the infection originates in the middle ear, the orbit becomes invaded by a circuitous route. Prompt measures, however, directed to the pathology in and about the ear, and drainage of the orbital abscess has effected cures in a fair percentage of cases.

The sphenoethmoidal infections offer an even better prognosis. Referring again to Eagleton's cases, 28 per cent arose from these two sources—surely a goodly number upon which to work when the serious prognosis of cavernous sinus thrombosis is considered. In these cases, the field of surgery is a broad one and calls for immediate cooperation and prompt action on the part of each. It is pertinent to add that, altho this is a form of disease which is not so common as other forms which we have discussed, in its management we are dealing with life or death, rather than with visual loss. Furthermore, by combined effort, it is probable that the mortality can be lowered to a considerable extent.

In calling attention to some of the important points of contact in a common field of surgery, I have endeavored to stress the need for, first, a frank and open discussion of an individual case from each of our several viewpoints; second, tolerance and respect for the opinions of each with a view to the best ultimate interest of the patient; and third, a plea for prompt surgical intervention when surgery is definitely indicated by the ocular and nasal pathology.

CHANGES IN THE REFRACTION OF THE EYE.

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The examples of defective ocular refraction first noticed, were of high degree, were considered very exceptional, were generally known to have existed for years, if not thruout life, and were not fully understood or accurately measured. So that changes in the defect of a moderate amount would be wholly overlooked. There naturally grew up an impression that such defects were individual peculiarities, like the stature, the color of eyes and hair, the features of the face. They were thought of as rare anomalies, and supposed, like other peculiarities of bodily makeup, not to undergo essential change thruout life. This impression was strengthened by the observation and record of other cases, each with its peculiarities, that did not change during the brief period they had been observed.

Even the understanding of accommodation did not reveal the changes that occur in refraction of the eye. The effects of accommodation were confused with myopia. The fact that hyperopia might be concealed by accommodation was held to account for increase of hyperopia in later life. The difficulty of measuring astigmatism while accommodation was present was a chief argument for the use of cycloplegia in the measurement of ametropia. The belief in the permanence of errors of refraction was easily maintained in the face of evidence to the contrary. If a patient came wearing lenses that obviously did not correct his ametropia, the easy explanation was that they had been given after an inaccurate measurement. If the prescriber did not use cycloplegics, and the critic believed them necessary, there was sufficient reason to ascribe the inaccurate correction to faulty diagnosis. If glasses were prescribed after cycloplegia, and the critic did not believe in the measurement of refraction "under the influence of drugs," the discrepancy was again an evidence of faulty diagnosis. Partisanship hid the scientific facts, and the debate as to whether it was better to use cycloplegics or not went merrily on.

There has been such diversity of methods used in measuring refraction, from the exposure of stock lenses and letting the customer choose what he liked to setting the patient down before an ophthalmometer and reasoning out what lenses he ought to need, and patients have tolerated such imperfect corrections of their ametropia, that data with regard to changes in the refraction which inspire confidence in their accuracy have been scanty. Even when the same methods of measurements have been employed, and by the same observer, he has not always felt confidence in the accuracy of his earlier findings when a later testing showed a different result.

A glance at the history of our knowledge of errors of refraction will help understand the subject before us. Nero, watching the combats of the gladiators thru his emerald, was pretty certainly a case of myopia corrected by a concave lens. The suggestion of a Roman writer, that all emeralds of that shape should be reserved for such use, seems to indicate an understanding of its importance. But after that, little more seems to have been known about myopia until, in 1601, it was studied by Kepler, who was myopic. His investigations were thought, by Donders, to have laid the foundation of physiologic optics.

Presbyopia and its correction by convex lenses were fairly understood and widely utilized from the time of the introduction of spectacles in the thirteenth century. But hyperopia was not recognized until much later. In 1772, Janin, of Avignon, mentioned a defect of the eye in which neither near nor distant objects could be seen distinctly without a convex lens, as after a cataract operation. In 1811, Wells reported that, at 55, he needed a 36 inch convex lens to see distinctly at a distance. In 1812, James Ware called attention to young persons, "for whom a glass of considerable convexity is required to enable them to see distinctly not only near objects, but also those that are distant." But the scattered observations of this kind attracted little importance to the defect. Its significance was better understood by Reute, who, in 1853, described it under the name "oversightedness." Two years later, Stellwag von Carion called it "hyperpresbyopia," a name rather widely adopted, until Donders described it in 1860, and more fully in his "Accommodation and Refraction of the Eye" in 1864.

Astigmatism was discovered still more recently. Thomas Young found in 1793, in his own eyes, what he called normal astigmatism, ascribing it to the crystalline lens. That astigmatism

might be a cause of serious impairment of vision, was first pointed out by Sir Charles B. Airy, Professor of Mathematics in Cambridge University, in 1827. He had compound myopic astigmatism, requiring for its correction a concave cylinder of about 8 inches focus, axis 145 degrees. He first noticed that he did not see with his left eye, and later noticed the elliptical diffusion image with its major axis at 35 degrees from the vertical. An optician named Fuller ground a compound cylindrical lens for him, with which he could read the finest print. In 1843, Dr. Isaac Hays, of Philadelphia, published in the American edition of Lawrence's Treatise on Diseases of the Eye, an account prepared by the patient, a clergyman of central New York, regarding his own eyes, from which account, McAllister, a Philadelphia optician, had prepared cylindrical lenses which "much improved vision." In 1848, Dr. Goode, who studied at Cambridge, reported his own astigmatism, of about 3. D., and that there were three other gentlemen at Cambridge who had a similar defect. In 1849, Professor Stokes, who had followed Airy as Professor of Mathematics, described the "Stokes lens" for the diagnosis of astigmatism. Whewell had proposed for this defect the name "astigmatism." In 1859, Knapp wrote on the curvature of the cornea. But it was the work of Donders, culminating in his "Accommodation and Refraction of the Eye" in 1864, that established the common occurrence of astigmatism, and brought appreciation of its importance.

It had to be established that myopia, hyperopia and astigmatism existed apart from accommodation and presbyopia, and were common and important enough to be worthy of general study on the part of the medical profession, before they could be widely enough and long enough studied to determine that they did change, and what these changes commonly were and their practical importance. They have now been studied in enough eyes, by enough observers, and over long enough periods of time, to make the recording and discussion of our observations interesting, profitable and of practical importance to our patients and to ourselves.

Now that we know such changes occur, and that they are important enough to demand attention, they may be considered from several different standpoints. Their exact nature, location, causes, rate of progress, and continuance; their relation to age, use of eyes, ocular disease, and to general health and nutrition, are all to be inquired into. But with our present knowledge and

lack of knowledge, a clinical and statistic study of the writer's own experience may be helpful in suggesting lines along which future studies may be carried on. It may suggest a classification of data that will facilitate further investigations regarding such changes.

My case records include 729 cases seen in private practice, in which the state of refraction was tested and recorded over a period of ten years or longer. These have been arranged, according to the age at which the ocular refraction was first measured, in four groups: (A) before 20 years of age; (B) between 20 and 40; (C) between 40 and 60; (D) over 60 years. In each of these groups, the different forms of ametropia were segregated, as myopia, hyperopia, and regular astigmatism.

A few cases illustrating some of the most striking changes can be mentioned in some detail; and some conclusions as to general tendencies of change, and probable mechanism of the change, are suggested. Only cases were included that have been studied in the last 20 years. In this way, a certain uniformity of technic was secured, which laboratory workers have found excludes many errors that are likely to creep in when the observations of various workers, or measurements by different methods, are compared. The age groups are first considered, and later the different forms of ametropia and kinds of change found.

In the period before twenty years of age, the cases of high ametropia of congenital origin are prominent. But such cases were found in all the later groups. In general, there is a great overlapping of age periods apparent in these groups; and actual changes to be expected at different periods of life could best be brought out by comparison of all patients who have been watched in each ten-year period. Among the 123 first seen before 20, were 98 who were followed after 40; and among 356 first seen between 20 and 40, were 310 seen after 40, and 10 examined after 60. Of the 241 first measured between 40 and 60 years old, 119 were also seen after they were 60. From such an incomplete and confused analysis of the cases studied, it is not possible to bring out many of the facts of changes in ocular refraction, but if we can learn that they occur, their frequency at different parts of life, and their practical importance, *particularly in later life*, the time will not have been wasted.

Age Groups.

The cases first seen before twenty years of age include some

in whom the ametropia was probably congenital. But as the patients when first seen were none of them less than two years old, the decision as to whether the error of refraction existed from birth or had developed subsequently, could only be reached with probable correctness upon collateral evidence. The same sort of evidence would be available with regard to such cases seen later in life. It is rather outside our subject to discuss them here, and the number that probably belong in this class is too small to furnish a basis for a statistic study. The impression made by going over the series of cases is, that such cases probably are less likely to change than cases in which ametropia has developed after birth, except the cases in which the ametropia is associated with partial, or complete, luxation of the lens.

The relations of the crystalline lens with its capsule and the ciliary body are known to develop in later fetal life, and are supposed to determine, to some extent, the refraction of the eye. It has been supposed, that tendencies of prenatal development, carried over into infancy, might influence changes in the refraction during infancy. It is well known, that almost all eyes are hyperopic at birth, that some become less so during childhood, and that some pass over into myopia. Is any general tendency in this direction indicated by these statistics? In Group A, the ages when the refraction was first measured varied from 2 to 20 years, the average being 9 years. When last measured, the ages varied from 13 to 39 years, averaging 26 years. It should be noticed, that this average age at which the hyperopes were first examined was 8 years, while the myopes were first seen at 13 years. This points to myopia, as a notable defect, appearing after the children had been subjected to the unfavorable conditions of school life.

Summary of Statistcs.

In summing up the changes in refraction found in this series of cases: When the record showed changes of 0.25 D., or less, it was set down as "no change." When the change was more than 0.25 D., but less than 1 D., it was set down as "slight." When it was 1 D., or more than 1 D. it was counted as a decided change in refraction. Among the 123 cases first examined before 20 years of age, there were 34 cases of myopia. Of these, 3 showed no change of amount, 7 slight increase, and 12 increases of 1 D. or more. There were 7 that showed slight decrease, and 2 decreases of 1 D. or more. Among 89 cases of hyperopia, 25 showed no change of amount, 31 slight decreases, and 7 decreases

of 1 D. or more. There were twenty that showed slight increase of hyperopia, and 9 increases of 1 D. or more. That is, among the 123 cases, there were 28 that showed no change of refraction, 38 slight increases, and 19 decided increases—higher M or lower H. But there were also 9 that showed decreased myopia, 7 slight, 2 over 1 D., and 29 that showed increased hyperopia, 20 slight and 9 1 D., or more. Thus, among the 123 cases, 57, or 46 per cent, showed increasing, and 38, or 31 per cent, decreasing refraction.

As to changes in astigmatism: 38 showed no changes in the amount. It increased in 62, slight in 45, and 17 decided. It decreased in 14, slightly in 9, and decidedly in 5. As to changes in the direction of the principal meridians, changes of 10 degrees or less were noted as "no change." This was the condition in 46 cases. But in 35, or 28 per cent, there was a change of more than 10 degrees. In 8 cases, 23 per cent, the meridian of greatest curvature shifted *toward the vertical* (astigmatism with the rule); and in 27 cases, 77 per cent, it shifted *from the vertical* (against the rule, inverse astigmatism).

These details have been dwelt upon to illustrate the method of this statistic study. Its main results in the other groups, B, (from 20 to 40 years), C, (from 40 to 60 years), and D, (over 60 years), can be stated more briefly, and all will be published in tabular form for easy comparison.

In Group B, there were 356 cases, of which 94 were myopes. Of these, 9 showed no change of myopia, 38 showed slight, and 11 greater increase of myopia; and 32 showed slight and 4 greater decrease of myopia. There were 262 hyperopes, of whom 25 showed no change, 78 slight and 12 decided decrease of hyperopia. As to spherical refraction, the whole group had 34 that showed no change, 139 increased, and 190 decreased refraction. The amount of astigmatism did not change in 94 cases. There were 130 whose astigmatism increased slightly, and 24 decidedly, making 154 with increase. There were 78 in which the astigmatism decreased slightly, and 12 decidedly, 90 of decrease. The meridians remained unchanged in 135 cases. There was change of over 10 degrees, in meridians of greatest curvature *toward* the vertical in 9, and *from* the vertical in 198 cases.

In Group C, first seen when 40 to 60 years of age, there were 214 cases. Of these, 49 were myopes, six showed no change of spherical refraction, 14 showed slight and 11 greater increases of the myopia, and 13 slight and 5 greater decreases of myopia.

There were 175 hyperopes, of whom 18 showed no change of refraction, 54 slight and 17 greater decrease of the hyperopia; and 64 showed slight and 15 greater increase of hyperopia. This makes 104 with increased, and 97 with decreased refraction. The amount of astigmatism did not change in 54 cases. It increased slightly in 81, and decidedly in 24, a total of 105. It decreased slightly in 38 cases, and decidedly in 5, a total of 43. In 44 the meridian remained unchanged, in 3 the greater curvature slightly shifted toward the vertical, and in 148 from the vertical.

In Group D, first seen after 60 years of age, there were 36 patients. Of the 5 myopes, in one there was no change in amount, one increased slightly and two decidedly, and one decreased slightly. There were 31 hyperopes, of whom 4 did not change in amount, 8 increased slightly and one decidedly, while 14 decreased slightly and 4 decidedly. The change in spherical refraction, therefore, was increase in 21 and decrease in 10. In only 7 did the change amount to 1 D.

In the statistics of a large number of cases, observed over rather long terms of years, the figures summarizing those observations should bring out the general tendencies in the changes of ocular refraction. Changes of certain kinds and forms will need to be worked up in special studies. The important changes that bring about myopia and keep it progressive, and the changes that make it regressive, are of such special character. They have been recently considered in a paper not yet published. It has been well known that myopia arises in early life, and the influence of its beginning is apparent in Group A, first examined before the age of 20. But the influence of age on the spherical refraction of the eye may be a general influence that must be noticed here.

It is significant, that in group A there were fully 47 per cent that showed increase of refraction, increasing myopia or decreasing hyperopia, and 31 per cent that showed decreased refraction, increased hyperopia or decreased myopia, while in 22 per cent no notable change of refraction occurred. In Group B, there were 356 cases, of which only 34 showed no change, and 110 change of less than 1D. In the whole group there were 139, or 39 per cent, that showed increased and 190, or 53 per cent, that showed decreased refraction. This is a larger percentage of change and a much larger per cent of diminished refraction, increased hyperopia.

In Group C, first seen between 40 and 60, the change in these percentages is still more notable. Among the 214 cases, there were 96 cases, 45 — per cent, of increased and 97 cases, 45 + per cent, of diminished refraction, while less than 10 per cent showed no change.

In Group D, first seen after 60, there were only 5 who did not change in spherical refraction, which increased in 2 cases and decreased in only 10, or percentages of 58 and 28. Comparison of these groups shows in early life an increasing tendency to change in refraction with increased hyperopia, but after 50 an increasing number in whom the hyperopia is decreased, or myopia increased, the "second sight" of age, which has long been known, both in medical literature and popular tradition.

With reference to regular astigmatism, these statistics reveal still more striking and important changes. In Group A (123 patients) under 20, astigmatism increased in amount, slightly in 45, decidedly in 17, a total of 62. It decreased in 14, slightly in 9, decidedly in 5. Before 20 there are changes in amount of astigmatism generally in the direction of increase. In Group B (356 patients), there were 130 whose astigmatism increased slightly and 24 decidedly, 154 with increase. There were 90 cases of decrease, 12 decided (1 D. or more), 78 slight (less than 1 D.). From 20 to 40, the tendency to increase of astigmatism continues but is less preponderant. From 40 to 60 (214 cases), the amount of astigmatism increased slightly in 81 and decidedly in 24, total 105. It decreased in 43, decidedly in 5 and slightly in 38. In patients first seen after 60 years of age (36 patients), the astigmatism increased in amount in 20 and decreased in 16. At all ages the amount of regular astigmatism is likely to change, altho in the majority of cases such changes are relatively slight.

The most striking changes in refraction are the changes in the directions of the principal meridians of astigmatism. These changes are not merely apparent, from inaccurate determination of the principal meridians, altho they are likely to be overlooked on that account, and they increase in number and importance with advancing age. At all ages the tendency of the change in the meridians is for the meridian of greatest refraction (axis of the convex cylinder) to shift from the vertical, what has been called "astigmatism with the rule," toward the horizontal, long called "astigmatism against the rule," which is the more common form in advanced age. This change in astigmatism has

gradually become more widely appreciated by ophthalmologists in the last 30 years, but the full force and importance of it had not impressed me until I studied these statistics.

In preparing these statistics, no cases were counted as having a change of meridians unless the change was over 10 degrees. Among 35 cases in Group A, first seen before 20, the meridian of greatest refraction had shifted *toward* 90° in 8, or 23 per cent. It had shifted *from* 90° in 23, or 77 per cent of those in whom a change was counted. Among 207 cases of Group B, between 20 and 40, the meridian of greatest refraction had shifted *toward* 90° in 9, and *from* 90° in 198, or 4 percent and 96 percent. In Group C (214 cases), 40 to 60, the meridian had not shifted more than 10 degrees in 44 cases; it had shifted toward 90° in 3 cases, and from 90 degrees in 148, percentages among those that shifted, of 2 and 98. The group of 36 patients seen after 60 showed almost wholly the results of such changes rather than actual changes during the concluding years of life.

CHANGES IN REFRACTION IN 729 PATIENTS

	A	B	C	D
SPHERIC REFRACTION				
Total Cases	123	356	214	36
No Change	28	34	24	4
Increase M.	19	49	25	3
Decrease M.	9	36	18	1
Decrease H.	38	90	71	18
Increase H.	29	154	79	9
Increased Refraction	57	139	104	21
Decreased Refraction	38	190	97	10
ASTIGMATISM				
Total Cases	114	338	202	35
Increase	62	154	105	15
Decrease	9	90	43	13
No Change of Axis	38	94	44	1
From 90°	27	198	148	32
Toward 90°	8	9	3	2

The exact anatomic changes in the eye bringing these changes in refraction are still a subject for special study, altho a few loose inferences regarding them may be helpful in pointing the way for further investigations. In childhood, youth and early adult life, there is some indication that the change is one of increased anteroposterior axis. This is certainly true for acquired and progressive myopia; but does not receive much support from statistics of hyperopia. As to the changes that occur in later life, the ophthalmometric studies of the cornea very rarely show

changes in its curvatures, and the changes of astigmatism cannot be of axial origin. We are forced to believe that these changes are chiefly, almost wholly, lenticular. Since Priestley Smith observed the increased size of the normal crystalline lens from 25 to 65 years of age, and suggested this was probably the cause of the increasing hyperopia of later life, no more probable hypothesis has been offered. These statistics show that the changes in shape of the crystalline lens must be studied anew, with new exactness and by new methods, and that such studies will greatly increase our knowledge of its physiologic and pathologic alterations. It is too large a subject to enter upon its discussion at the end of this paper.

A few outlines of cases, the more recent or striking, among hundreds of more or less similar cases, may illustrate the character of this investigation and the enormous practical importance of these common changes in refraction.

A boy of 15 was wearing glasses for myopia; R., —3.50, L., — 3.75. These had been changed repeatedly, and the vision continued to grow worse. He came because it was now reduced to 0.7. His correction, R., — 2.87 ⌒ — 1.25 Cyl. axis 95°. L., — 2.50 ⌒ — 0.75 cyl. ax. 85°, gave vision 1.2 and seems to have stopped the progress of his myopia.

A woman of 47 required R., — 1. with — 1.25 cyl., axis 5°, and L., — 1.25 with — 1. cyl. axis 5°, and + 2.50 added for near. After four changes of glasses she now requires R., — 5.5, L., — 2.25 Sph. ⌒ — 1.75 C. axis 23°. Lens opacities have been visible for 12 years, but, with vision 0.6, the eyes are equal to the reasonable demands made upon them.

A lawyer, aged 59, had glasses from a careful oculist 16 years before, who said, that for distance they would not need to be changed again. The deficiencies on account of presbyopia had been made good by opticians, until + 3.25 had been added for that purpose. The correction of the present hyperopic astigmatism with + 2.50 added for near, gave relief from the headache, stiff neck and other nervous symptoms, complained of.

A business man of 73, had worn glasses, obtained from an optometrist, for 20 years. These had been frequently changed and he now wore R., — 0.75, L., — 0.50 sph., with + 2. sph. added for near. He went back to have them changed, and was told by the optometrists that they could do nothing more for his eyes, in the way of glasses. His correcting glasses, R., — 0.75 cyl. ax. 87°, L., + 0.50 sph. ⌒ — 1. cy. axis 95°, gave him

vision 1.2 in each eye; and with + 2.75 added for near, his eyes seemed and felt as good as ever.

A woman came to me first at the age of 43. Last week, at the age of 75, she returned wearing the glasses I had given her 10 years ago. Her eyes felt weak when she read in the evening. She thought it was time to have her glasses changed, her vision with them being 0.7. The distance correction required + 0.75 more spherical, and + 0.50 and + 0.25 more cylinder. With these, and + 2.75 added for near, she can read freely. Putting on the new glasses and walking a few blocks to show them to me, she had already noticed that her eyes felt rested by them.

A woman of 47, with some signs of arteriosclerosis and high blood pressure, came three years later with some lens haze and cortical striae, vision being reduced to R., 1, L., 0.9. After this, her glasses were changed at intervals of 3 and 2 years, and after 12 years, she still has vision with lenses of 0.7 in each eye, and can use them freely for distance and reading.

A woman at 59 required R., + 2., L. + 2.25 sph. With 2.50 added for near, vision, 1 plus. Nine years later, there were faint lines of opacity in the lens cortex, and her eyes were less hyperopic. The hyperopia passed over into myopia, which at the age of 72 had increased, requiring R., — 4.50 sph., L., — 1. sph. with — 1 cyl. axis 10°. With these she had vision of 0.7. At 75 she required R. and L. — 7. sph., which gave vision of 0.3 in each eye. Soon after this, she had diabetes. At 76, cataract operation in the right eye gave vision of 0.8 with + 10.50 sph. ⊃ + 2.50 cyl. axis 10°. She died at 79 of acute disease.

A woman, aged 39, was first examined for glasses, being then almost emmetropic, but requiring R. and L. + 2.25 for reading. Since then, her lenses have been changed at rather long intervals until she required R., + 1.25 ⊃ — 0.75 cyl. axis 145°, L., + 1.75 ⊃ — 1. cyl. axis 35°. With these her vision is still 0.5, altho 20 years ago she came for blurring of sight and inability to recognize people, and showed haze and irregular refraction of each crystalline lens, which has slightly increased since. But she is still able to keep house, attend to her garden and recognize people, at the age of 87.

Someone may remember the satire of Dr. Johnson on the scientists of his time:

"Some of them spend their lives registering the changes of the wind; and die firmly convinced that the wind is changeable."

But that satire lost its point since the "Spirit of St. Louis"

took its nonstop flight across the North Atlantic, and since Commander Byrd sailed away for two years of observation of the winds of the Antarctic waste, while those who have arrived on the Zeppelin are pointing out that the need of aerial navigation is for a better knowledge of the air currents.

We now know that the refraction of the eye changes. This opens up new fields for thought and achievement. Such changes become more common, more prominent, more harmful in later life, when there is less accommodation to overcome them, and when the overcoming of their effects is more important for good vision. Is there any reason why the measurement of refractive defects in elderly people should be left to those whose interest in the scientific correction of defective refraction is subordinate to their interest in selling spectacles?

RECENT DEVELOPMENTS IN THE DIAGNOSIS OF INTRACRANIAL LESIONS, WITH SPECIAL REFERENCE TO THE VISUAL PATHWAYS.

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OMAHA, NEBRASKA.

That brain tumor is of frequent occurrence is a fact that should become more fully realized by the rank and file of medical practitioners, into whose hands the sufferers with this serious malady usually first come for relief. The general symptoms of this affection, nausea, vomiting, papilledema, etc., result, as is well known, from an increase of intracranial pressure, and constitute a well marked syndrome, generally not especially difficult to recognize by the alert physician. When the presence of such a lesion is evident, the question of localization becomes paramount, since correct localization is indispensible to whatever basic treatment may be possible, and in the absence of such treatment in progressive lesions of this kind, the outlook for the patient is hopeless.

Newer diagnostic methods, such as roentgen ray, ventriculography, etc., which throw light upon the problem of localization, are of the utmost importance. The development of these methods is watched keenly and hopefully by those whose task it is to diagnose and to treat intracranial lesions.

However, we must keep in mind that the possibility of progress by the older methods of localization is far from being exhausted. The most important older method consists in localization by a study of the loss, or modification, of function in the socalled neural projection systems, which are the pathways of communication between the central nervous system and peripheral tissues of the body. It is reasonable to expect progress by this method until our more or less imperfect present knowledge of the anatomy, physiology, and psychopathology of these pathways is rendered satisfactory by means of fundamental research.

Considered as a whole, there are two neural projection systems, corresponding to the two divisions, somatic and visceral, into which the tissues of the body are differentiated. Since each

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of these two great tissue systems which compose the body has an inward and outward connection with the central nervous system, the two main peripheral neural systems are correspondingly divided into afferent, or sensory, and efferent, or motor (or secretory) components, thus giving us in all four subdivisions of the peripheral nervous system. These are generally classified, as is well known, as the somatic afferent and efferent, and the visceral afferent and efferent components, which therefore include all the fibers composing the peripheral nervous system. Gaskell first made a detailed study of the nerve fibers to visceral and vascular tissues. The clarification of our knowledge of the structure and organization of the peripheral nerves, especially the functional components of the cranial nerves, is due mostly to American investigators, such as Herrick, Strong, Johnston, Coghill, Norris, and Willard.

These four socalled nerve components are themselves each composite, and there is not a little yet to learn of their anatomy, physiology and higher functions. As yet, we are able to obtain relatively little localizing data from a study of the visceral afferent and efferent neural lines of communication. It is true that there may be a disturbance of cardiac rate and rhythm as general or pressure results of brain tumor, and such symptoms tend to be earlier and more severe in lesions of the posterior cranial fossa. Yet on the whole, aside from the special visceral afferent senses of taste and smell, a study of the visceral lines of communication yields in the present state of our knowledge relatively little information of intracranial localizing value. It is possible that further study in this field may yield useful localizing data.

On the other hand, we understand much about localization of intracranial lesions from information obtained by a study of disturbances, or defect, of the somatic afferent and efferent divisions of the peripheral nervous system and their correlative cortical centers. In fact, the science and art of cinical neurology has been until recent years largely built upon the study of the somatic components of the nervous system. This holds true for both the somatic afferent (sensory) and the efferent (motor) components. The study of these systems has yielded a great deal of data of more or less exact localizing value. But even in the somatic projection components, fundamental biology has much yet to discover as a basis for clinical advance. Histoneurologists are well aware that miscroscopic study of the peripheral nervous system reveals many nerve fibers of yet undetermined functions, and moreover,

our knowledge of the cortical projection centers and association pathways are far from satisfactory. Our lack of exact knowledge has been especially evident in the visual mechanism, the most highly specialized receptive unit of the somatic afferent system. Some recent advances in our knowledge of the internal organization of the visual apparatus will be reviewed here, as a possible means of advance in localization of intracranial lesions.

The intimacy of the visual apparatus with the central nervous system is correlated with the fact that the retinae themselves are only areas of the embryonic brain, thrust to the periphery to receive, by means of a delicate and highly complex photographic mechanism, images of the external world. The central pathways take a long intracranial course, partly immediately under, and partly thru, the brain substance to the occipital pole, where they project upon the cortex, and thence indirectly upon the higher cerebral centers. Moreover, the eyes have wide spread connections between the brain and spinal cord by means of afferent and efferent somatic and visceral nerves. On account of lack of space, intracranial localization from a study of disturbed functions of the ocular nerve elements—voluntary motor, general sensory and autonomic—will not be discussed in this paper, tho it is true, as noted above, that there is also much yet to learn of fundamental biologic importance in connection with the components of these nerves, and their central connections; nor shall a discussion be given of the higher visual cortical connections.

There has been considerable recent development in the problem of the organization and localization in the visual pathways. The ophthalmologist and clinical neurologist have long felt the need of more complete and definite knowledge of the arrangement and organization of the "primary" and "secondary" visual nerve fibers, their sites of reception, and their indirect connections and functions as regards the higher cerebral centers. This communication will be devoted to a brief discussion of some of the recent biologic and clinical advances that have developed in the field of the visual projection system.

As regards the first link in this system, interesting and important researches have been reported recently by Brouwer and Zeeman[1] upon the arrangement and organization of the fibers in the optic nerves, chiasm, tracts, and their endings in the lateral geniculate body. The animals used were the rabbit, cat, and ape. Their work upon the latter animal only will be here discussed somewhat in detail. The eye was drawn forward after the appli-

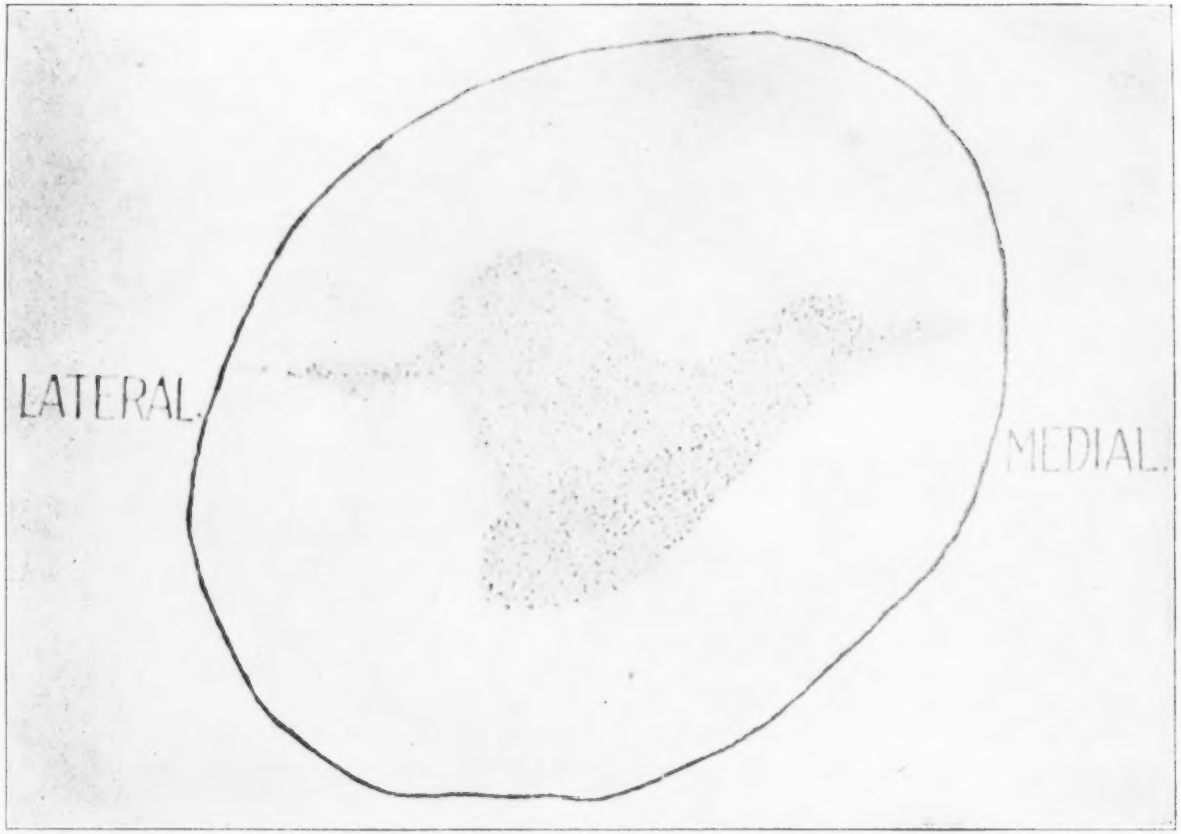

Fig. 1. Secondary degeneration in the optic nerve after a macular lesion. Especially noticeable is the large size of the maculopapillar bundle. The fibers from this bundle tend to spread among the fibers of the peripheral retinal quadrants. Brouwer and Zeeman, Brain XLIX, p. 15.

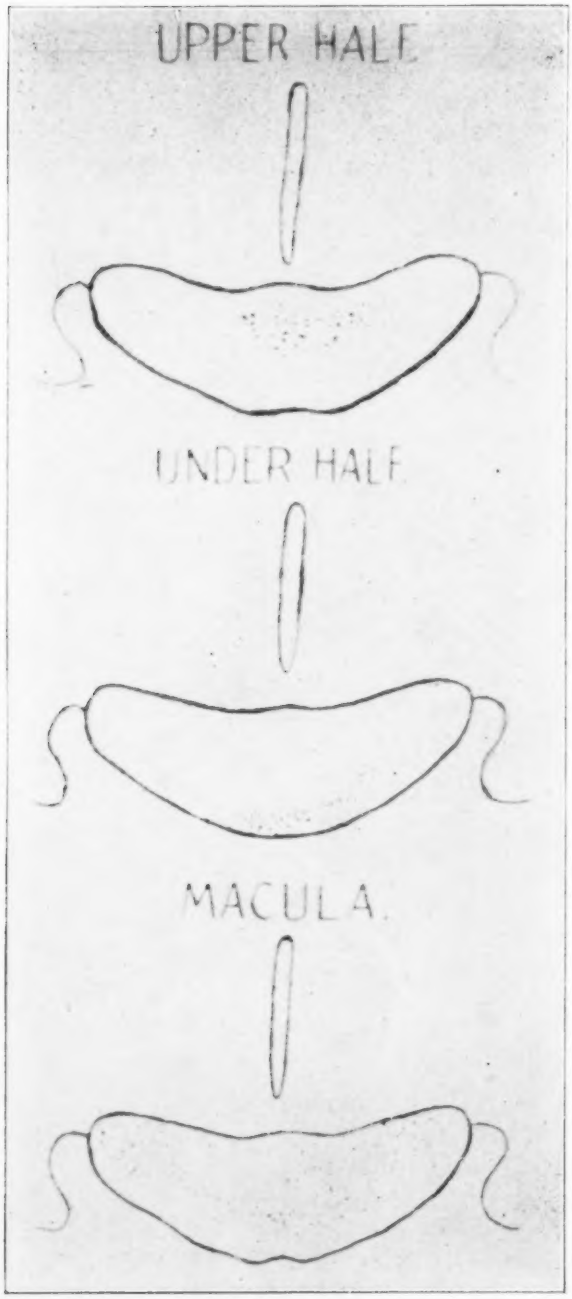

Fig. 2. Secondary degeneration in the crossing fibers in the chiasm after lesions in the upper and lower halves of the peripheral retina, and after a macular lesion. Brouwer and Zeeman, Brain XLIX, p. 24.

cation of cocain and atropin, and a long knife was passed thru the temporal conjunctiva, as directed by the aid of the ophthalmoscope, to localized areas of the peripheral quadrants and macula. Cirsumscribed lesions were made at various points in these areas, the animals were killed in eighteen days, and nerve fiber degeneration was traced by the Marchi method.

The authors found that the fibers from the peripheral retinal quadrants are fairly well localized in the optic nerves. The upper retinal fibers tend to lie above in the nerve, and the lower, below. Similarly, the nasal fibers tend to lie medially, and the temporal, laterally. The centrally projecting fibers from each retinal quadrant, therefore, tend to occupy in the optic nerves a position corresponding to the location of the retinal quadrant whence these fibers arise.

The maculopapillar bundle occupies a mid position in the nerve (Fig. 1). A striking feature is its relatively large size. Localization is not strict, since there is considerable overflow of macular fibers into those from the peripheral quadrants. Just back of the eyes, the macular bundle has a somewhat lateral position, where it separates the temporal fiber quadrants. Then it trends axially to occupy, toward the chiasma, a central position. The fibers from the upper part of the macula apparently lie above those from the lower.

In the chiasma, the fibers from the upper quadrants remain above, those from the lower, below. The crossing macular fibers occupy a mid position, and tend to avoid the chiasmal surfaces and borders (Fig. 2). The fibers from the upper half decussate somewhat later than those from the lower. It was not quite clear whether the upper macular fibers cross dorsally and the lower ventrally. Proximally in the chiasma, the macular fibers shift dorsolaterally to take the position which they occupy thruout the optic tracts.

The various parts of the retina are here represented in the chiasma as Henschen and Wilbrand found them in man. Brouwer[2] noted that the organization of the fibers in the chiasma explains why hypophyseal tumors cause upper quadrant field defects, since the upward pressing tumor impinges upon the lower surface of the chiasma, and therefore interrupts function in the lower or ventral retinal quadrants. There is clinical evidence, he says, that lesions pressing downwards on the chiasma similarly interrupt vision in the lower fields. The author in passing suggests this as a means of distinguishing between a hypophyseal or supra-

sellar tumor. His suggestion here correlates with the extensive research of de Schweinitz[3], Cushing[4], Benedict[5], and others in connection with visual defects caused by parachiasmal neoplamata.

The retinal fibers maintain a still somewhat different organization in the optic tracts. Here the fibers from the upper peripheral quadrants lie dorsomesially, and those from the lower, ventromesially. There is no overlap of the fibers from the peripheral retinal areas.

The macular fibers occupy a medial position between those of the peripheral quadrants, and widen laterally. On cross section, the macular fiber area occupies the entire lateral boundary of each tract, and gradually narrows, wedgewise, in a medioventral direction, separating the fibers from the upper and lower retinal regions. Medioventrally, the macular fibers overlap considerably those of the dorsal and ventral retinal areas. (Fig. 3.)

The organization of the tract into three principal fiber areas from macula and upper and lower retinal regions, foreshadows considerably the position of the sites of reception of these three fiber divisions in the primary optic centers, which will now be discussed.

The Primary Optic Reception Centers.

It is interesting to note, that in none of the experiments could primary optic fibers be found ending in the pulvinar of the thalamus. The center, therefore, should no longer be regarded as a primary reception station for retinal fibers. This fact coincides with the views of Henchen[6], who always held that hemianopsia is not produced by large lesions of the pulvinar which do not involve the lateral geniculate, but is produced by lesions of the latter which spare the pulvinar. The pulvinar, however, should not be regarded as having nothing to do with vision, since Minkowski[7] has shown that it has wide connections with the neopallium, especially the gyrus angularis, but also with the precuneus, and with some areas of the occipitoparietal cortex, and probably also with the occipitotemporal. The connection of the pulvinar with the angular gyrus suggests that it is concerned with higher visual functions, such as stereoscopic vision, and the recognition of absolute and relative distance.

The primary end stations of all retinal fibers were found to be corpus geniculatum exterum, and the corpus quadrigeminum anticum. In three only of Brouwer and Zeeman's[8, 1] experiments of partial lesions of the retina of the ape, were they able to follow

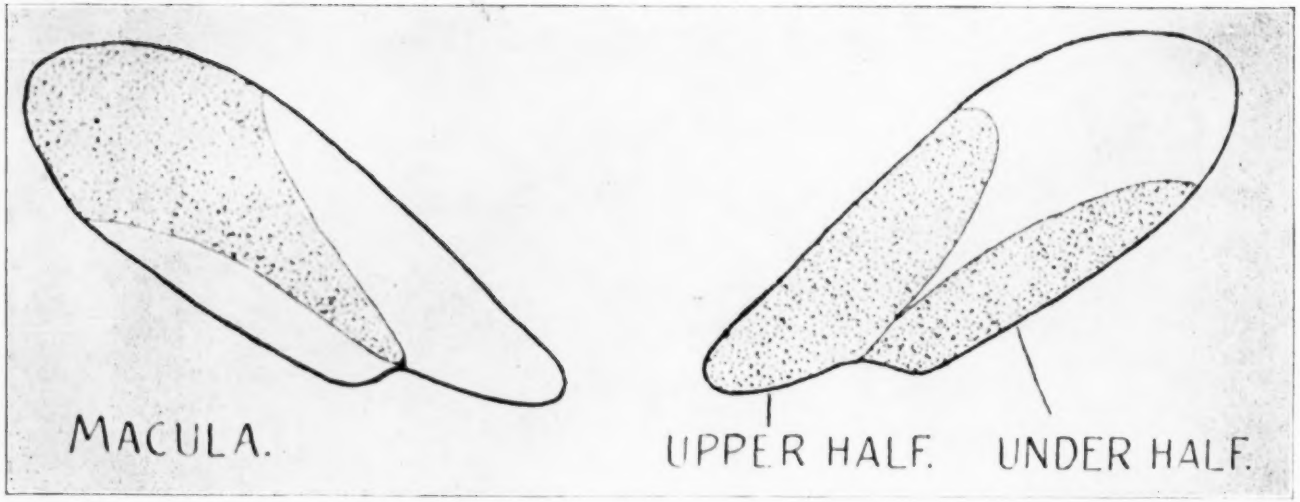

Fig. 3. Maximal secondary degeneration in the optic tracts after lesions in the macula, and in the upper and lower halves of the peripheral retina. Brouwer and Zeeman, Brain. p. 25.

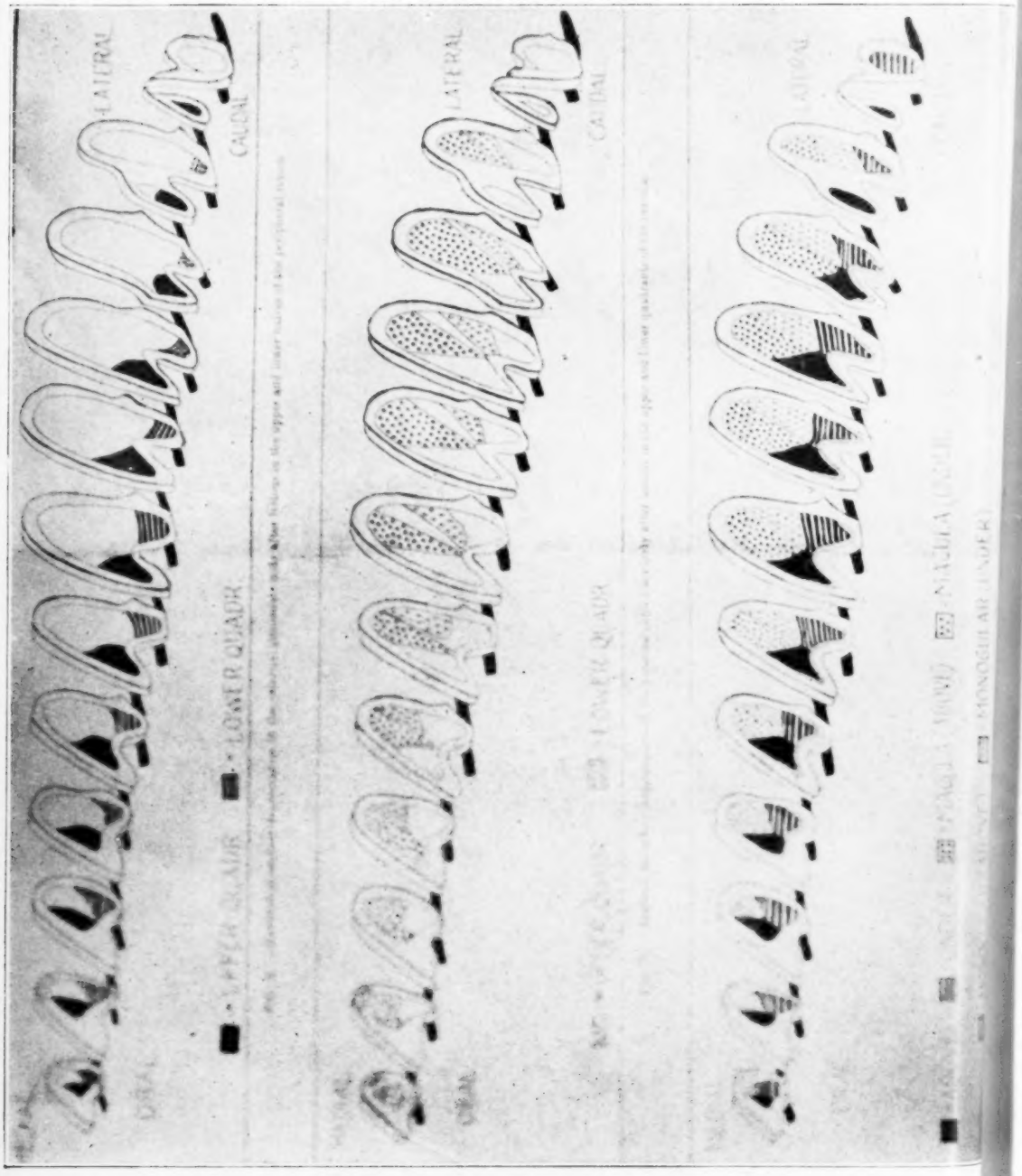

Fig. 4. The projection of the different parts of the retina in the dorsal nucleus of the external geniculate in the monkey. It will be noticed that the upper retinal quadrant fibers end medially (black), and the lower retinal quadrant fibers end laterally (striped); that the upper macular area (dotted) lies medial to the lower macular area (crosses); and that the monocular area lies in the narrow ventral margin. Brouwer and Zeeman, Brain, opp. p. 26.

degenerated fibers to their termination in the anterior quadrigeminal body. The sites of ending of these fibers differ from one another, a fact which suggests localization of the retina in the superior colliculus. The authors expected to find that degenerated fibers could be traced to the superior colliculus following macular lesions, since pupillary reactions are believed to be greater when light rays fall upon the macula. However, such fibers were absent, suggesting that if fibers for reflex pupillary movement thru this center arise in the macula, they must be non-myelinated. The retinal fibers are precisely organized in the superior colliculus of the rabbit and cat. In the ape, fewer fibers go to this part of the brain, especially on the same side, and the authors were unable to chart them.

Much the larger number of retinal fibers were projected upon the corpus geniculatum externum. Here a definite localization was found to exist. (Fig. 4.) The upper quadrant of the retina is projected medially in the dorsal nucleus, where it occupies a restricted area in the medial horn of the nucleus. The lower quadrant is projected to a restricted area in the lateral horn of the nucleus. These two areas do not overlap and are relatively small as compared with the macular reception site. Moreover, a localization of concentric zones of the different quadrants could not be differentiated in either of these two areas, since it could not be determined whether the partial experimental lesions had been located close to the macula or more peripherally in the same quadrant. Also, the crossed and uncrossed fibers must end close together in these receptive areas of the external geniculate bodies.

The genicular reception of the macular fibers, on the contrary, occupies a relatively large area, lying between and dorsal to the receptive sites for those of the retinal quadrants. It may be seen (Fig. 4) that the upper macular fibers (dotted) remain medial, and the lower macular fibers (marked with crosses) lateral. The upper and lower macular areas, therefore, lie respectively above and in contact with the areas for the reception of the adjacent peripheral retinal quadrants. A cross section of the lateral geniculate, therefore, shows that the upper retinal fibers are projected medially (black in figure). Just above and lateral is the upper macular area. Immediately lateral to this lies the area of the lower macula, which in turn lies in contact with and dorsomedially to the lower retinal area (striped). The figure shows also the narrow primary reception site for monocular vision located ventral to the binocular area.

It may be said here that the anatomic problem of the location of the retinal site of monocular vision is not yet solved.

The histologic structure of the lateral geniculate body has been studied by von Monakow[9] ('83), Cajal[10] ('04) and others. Excellent accounts of this primary visual reception station are given by Minkowski[7] ('13), Kappers[11] ('21) and Henschen[12] ('26). It has been shown by Minkowski, Kappers and others that the geniculatum externum consists mainly of two portions, a ventral and a dorsal. These are generally called the ventral and dorsal nuclei of the lateral geniculate.

Wollard[13] ('26) has recently studied this body in a series of closely related animals forming an ascending series. In tupia, which has monocular vision, and in which the retina shows no differentiated central area, the dorsal nucleus is small. In nycticebus, which has probably not a differentiated retinal area, the lateral geniculate body shows a highly specialized ventral nucleus, which Wollard relates to its nocturnal habits. Tarsius presents a highly differentiated retinal area, which may be considered a primordium macula lutea, and there is a corresponding development of the central mass of the dorsal nucleus. The marmoset possesses a differentiation of the central retina approximating the final form of the fovea centralis. The central area of the dorsal nucleus is correspondingly increased in size and differentiation. This study of Wollard's shows clearly that as the higher members of primates are studied, the ventral nucleus disappears, while at the same time there is an increasing elaboration of the dorsal nucleus running parallel with the specialization of the central retinal area.

Putnam[14], who has recently also reported a comparative study of the mammalian geniculostriate system, notes that the primitive type of corpus geniculatum externum consists of a ventral nucleus, without lamination, lying vertically on the surface of the brain stem. This is seen typically in marsupials and lower mammals. He notes that only the dorsal nucleus has a projection on the cerebral cortex. In his series, the cetacea, carnivora, and primates showed variations from the typical mammalian type of corpus geniculatum externum. There was the almost complete disappearance of the ventral nucleus, and certain deformations of the primitive form, perhaps resulting from an increase in size of the area of representation of binocular vision. There was a lateral rotation, so that the original external surface looks ventrally. The dorsal nucleus only, and not the ventral, atrophies after cortical lesions, which supports the view that the ventral nucleus is unconnected with the geniculostriate radiation. The ventral nucleus atrophies after section of the contralateral optic nerve, "but its connections are unknown."

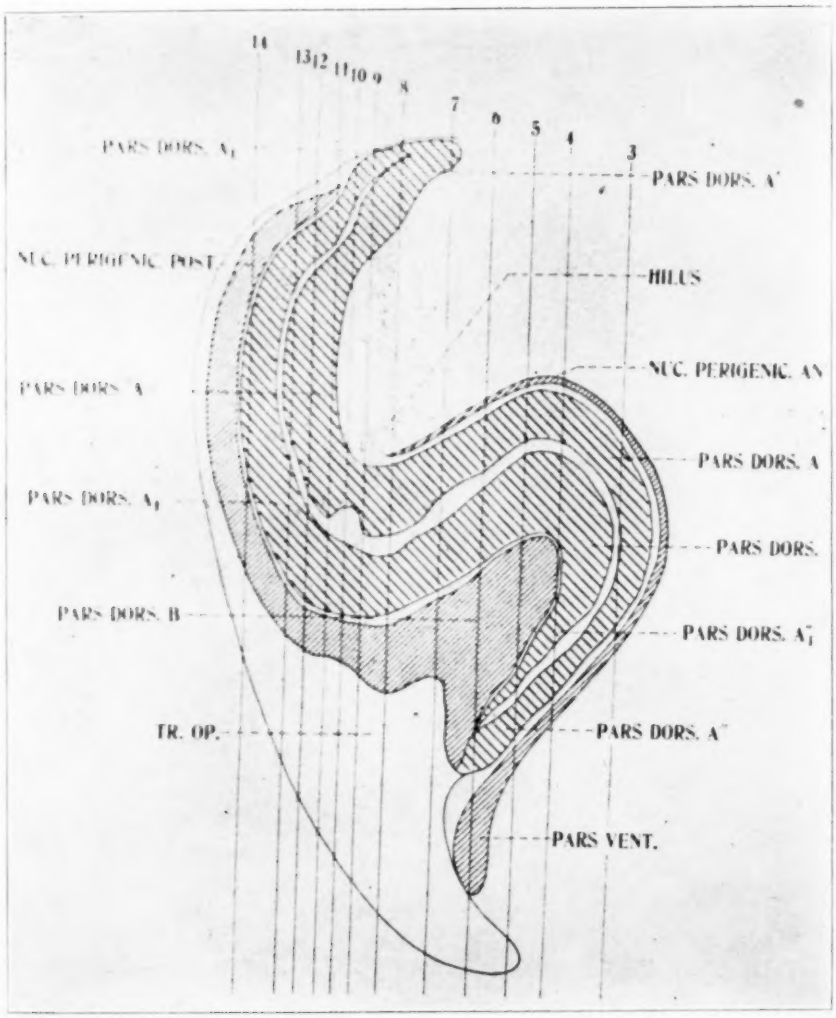

Fig. 5. Structurally, the lateral geniculate is composed as summarized by Thuma, of five distinct entities. 1. Pars dorsalis A and A_1 making up the main nucleus, forming together an S-shaped figure, and separated thruout by a medullary lamina. 2. A pars dorsalis B, ventral to the pars dorsalis A. 3. A pars ventralis, ventrolateral to the pars dorsalis and separated from it by a thin medullary lamina. 4. A nucleus perigeniculatus anterior, forming a thin shell over the pars dorsalis. 5. A nucleus perigeniculatus posterior forming a cap over the caudal pole of the pars dorsalis. For cytoarchitecture see Thuma's paper. The differential functions of these units in relation to the perception of form, color, movement, and the interpretation of distance, color vision, etc., nothing is yet known.

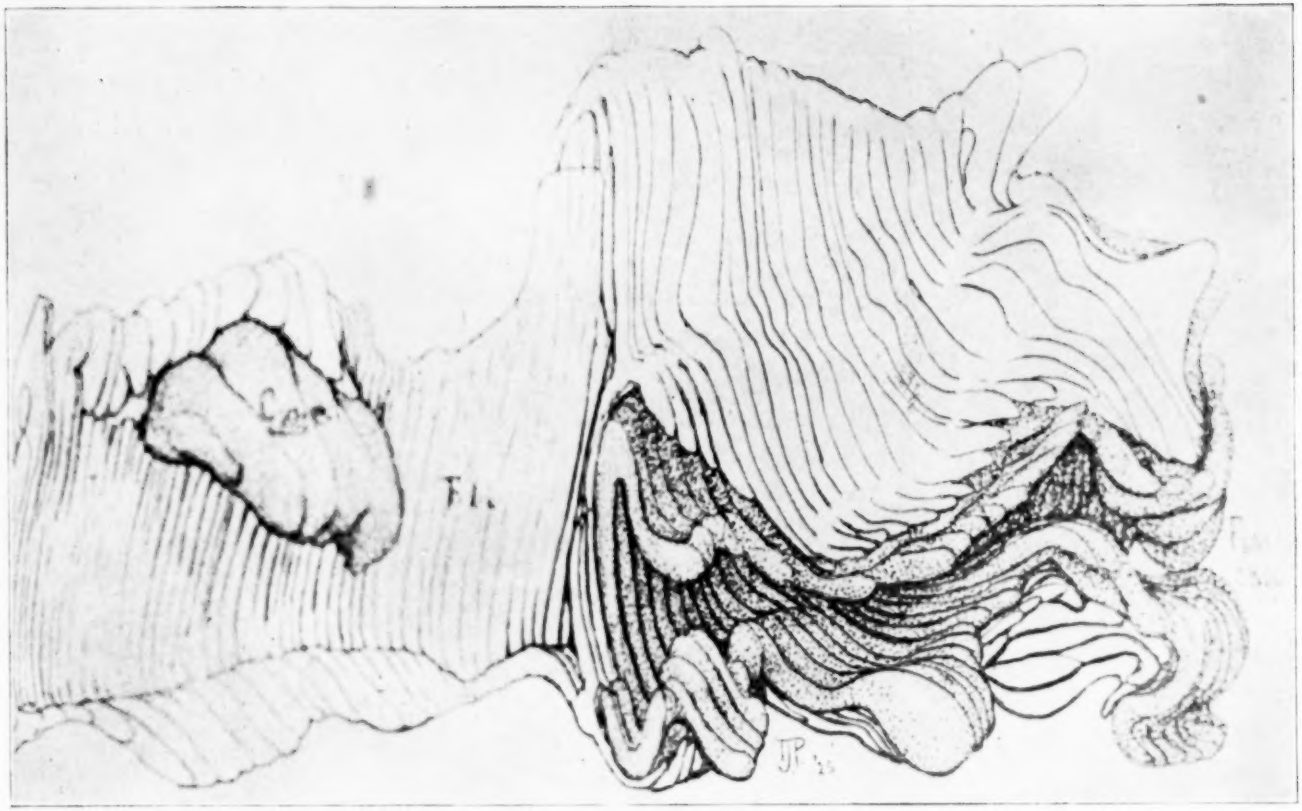

Fig. 6. Camera lucida drawing of wax plate reconstruction of the central visual system of a baby at term. Right hemisphere, viewed from the mesial side. The corpus geniculatum externum (C.g.e.) is heavily shaded. The fasciculus longitudinalis inferior (F.l.i.) is lightly shaded; the direction of its fibers is not indicated, but can in general be inferred from the shape of the model. The area in which fasciculocortical fibers lie is shown without shading or stippling. They are diffusely scattered thru the region outlined. The striate cortex is deeply shaded. Most of it lies in the depths of the calcarine fissure (Fiss. calc.). (Putnam IV.)

An idea of the histologic complexity of the corpus geniculatum externum in the carnivora (cat) may be obtained by an examination of the diagram of it as reconstructed recently by Thuma[15] in Huber's laboratory. (Fig. 5.)

The position of the macular area as given by Brouwer and Zeeman in the ape agrees almost exactly with its localization in man by Rönne,[16] who studied the ending of the maculopapillar bundle after its degeneration from toxic causes. This localization is further corroborated by the research of Wollard as noted above.

Two striking facts concerning the macular area in the lateral geniculate stand out here. One is that this region is relatively very large as compared with the extent of the corresponding peripheral area. This correlates with the relatively large area occupied by the macular bundle in the optic nerve and tract. The second fact is that there is a definite localization of the macula in the lateral geniculate. Both of these facts are significant in relation to the question of the extent and localization of the macular fibers in the central optic pathway and upon the occipital cortex.

The arrangement and organization of the optic fibers as described by the authors are on the whole considerably simpler and different at several points from the views formerly held, as may be illustrated by a comparison of figure 8, which shows an older scheme (Henschen's modified), as given in a recent publication by Traquair[17], ('27).

Brouwer[1] takes issue with Minkowski[7] upon the relation of the endings of the geniculate fibers from homonymous retinal points. Minkowski concluded that the crossed and uncrossed fibers come into receptive contact with cells of different genicular layers. His method was based on the principle of disuse chromatolysis of the genicular cells following sections of the optic nerve. Brouwer found the crossed and uncrossed fibers mixed. Thuma[15] has discussed this point in his recent analysis of the external geniculate in the cat, as noted above. He suggests that Minkowski's method may give a misleading picture, because genicular cells receiving afferent fibers from other than retinal sources would probably not show chromatolytic change upon optic nerve section, whereas those genicular cells receiving afferent fibers only from the retina would undergo chromatolysis. The complex lamination of the lateral geniculate body in the cat suggests an increase of nonoptic afferent stimuli, and these should function to maintain the morphologic integrity of certain of the genicular cells when the normal retinal influence is lost thru interruption of optic nerve fibers. As

Brouwer suggests, further research is required to clear up the divergencies of view resulting from these different methods, and to show conclusively whether the terminals of the crossed and uncrossed optic fibers intimately mix in the dorsal nucleus of the lateral geniculate body, or remain in separate laminae.

The workers in this field have utilized the principle that both retinal and cortical lesions cause regressive changes in the corpus geniculatum externum. Study of genicular degenerations after cortical lesions had shown that the different parts of the area striata were localized in the geniculate. But on account of the lack of definite knowledge of the retinal organization in this primary station of visual reception, the plan of retinal localization in the cortex could not be definitely deciphered.

The work of Brouwer and Zeeman and Rönne have clarified the problem of the organization of the primary optic neurons and their first reception stations in the ape and man. This information, obtained by purely anatomic methods, is especially valuable, since it furnishes the means, as Putnam[14] has shown, of integrating the known experimental facts concerning the central visual pathway, and especially has thrown light upon the question of the organization and localization of the retinal fovea and peripheral quadrants in the optic radiation and cerebral cortex. The older clinicopathologic studies had never been able to solve this problem satisfactorily. The weak link in the older methods has been considered the perimeter, which, tho clinically indispensable, yet leads at times to failure, even in expert hands, to locate scotomata, on the one hand, and to detect, on the other, islands of intact vision. The limitations of perimetry are seen, as Putnam notes, in the cases of Henschen and Lenz, which show that even careful and expert perimetry may miss large cortical defects.

The Optic Radiation.

The optic radiation arises from the dorsal nucleus of the lateral geniculate. None of the fibers arise from the ventral nucleus. The fibers spread out widely upon leaving the geniculate body to form the "stalk" of the radiation. Many of the fibers sweep forward to form the "temporal knee," and then extend dorsalwards to be distributed to the area striata. Figure 6 shows a wax plate reconstruction by Putnam of the optic radiation in a baby.

The course of the individual fibers are not shown in the figure. The radiation widens as it is traced toward the area striata,

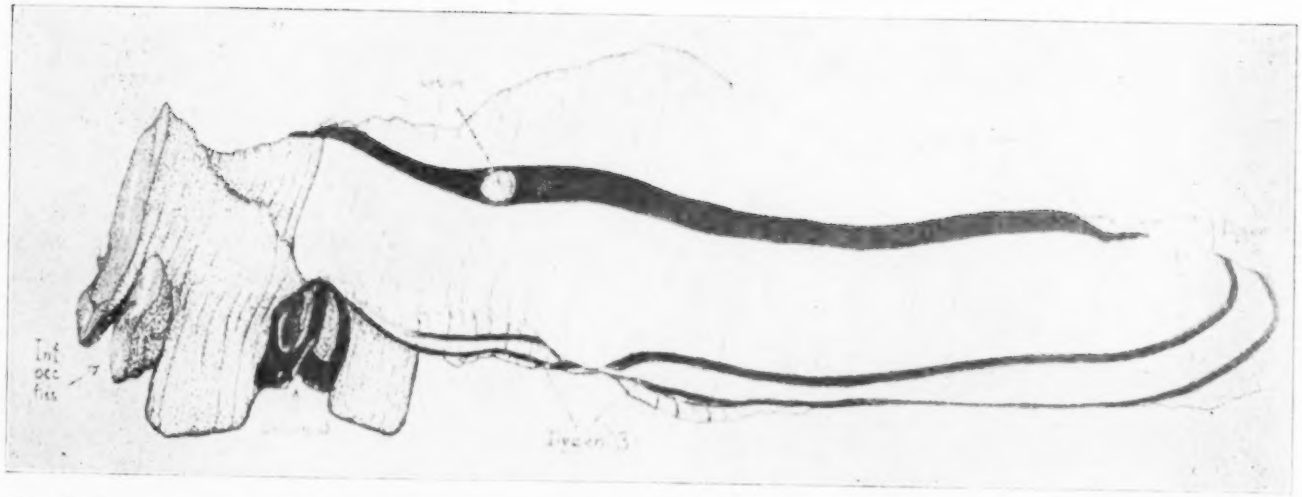

Fig. 7. Reconstruction by Putnam to show the course and position of the nerve fibers in the optic radiation in a case in which three small lesions caused streaks of degeneration in this part of the visual pathway. (IV.) The streaks of degeneration indicate the normal direction of the nerve fibers. At the right, these streaks converge upon the external geniculate body. At the left, lies the cerebral cortex. Inf. occ. fiss. indicates the inferior occipital fissure.

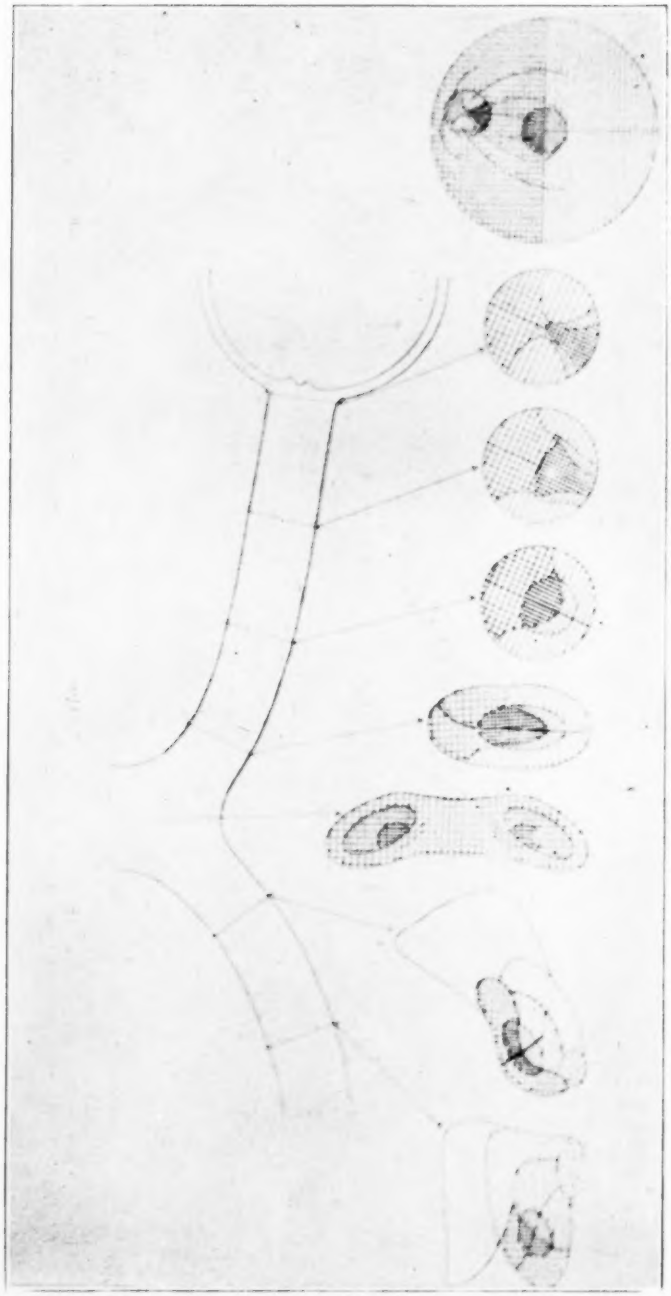

Fig. 8. Henschen's scheme (modified) of the distribution and course of the nerve fibers in the retina, optic nerve, and tract (right side). The chiasmal section is slightly modified from Wilbrand and Saenger. Traquair, An Introduction to Clinical Perimetry, 1927, p. 62.

and numerous fibers stream from its upper and lower lips toward the cortex. The fibers from the upper edge supply the striata area above the calcarine fissure, and those from the lower edge, the lower calcarine area. There was no evidence of crossing of fibers from the upper and lower calcarine lips as described by Pfeifer[18]. Behind the ventricle the radiation frays out into many scattered bundles, some of them supplying the part of the striate cortex extending upon the lateral surface.

Putnam's method of reconstruction is more accurate than that used by Pfeifer[18], as portrayed in the latter's recent excellent monograph. For three dimentional representation, the latter constructed a model of the gray matter of the cerebral cortex and basal gray masses. Into this he molded free-hand various conceivable forms of the optic radiation, and fitted them successively into the appropriate region of the concave model. The form which he judged to fit best into the space was one in which the fibers from the lower edge of the radiation extended to the anterior end of the calcarine fissure, and those from the upper edge, to the occipital pole. He supposes that the course of the macular fibers is in the dorsal part of the radiation. His scheme demands that the fibers originally dorsal supply the ventral calcarine lip, an arrangement which he favors but does not explain. This method is certainly much less accurate and dependable than the wax plate reconstruction from magnified histologic drawings as used by Putnam, since the latter is of necessity the exact counterpart in form and relations of the structure it represents.

The wax plate method of reconstruction has, however, the limitation that it does not represent in normal material the course and direction of the individual fibers. This information, however, was supplied from a reconstruction of the human geniculostriate radiation which presents small lesions that determined streaks of degeneration extending more or less thruout the entire length of the radiation from cortex to the corpus geniculatum externum. (Fig. 7.)

Is the Geniculostriate Radiation Identical With the Fasciculus Longitudinalis Inferior?

There has been much debate as to whether the optic radiation runs wholly or partly in the internal longitudinal fasciculus (stratum sagittale internum), or in the stratum sagittale externum (inferior longitudinal fasciculus). von Monakow[9] and his followers held that the optic radiation runs chiefly or entirely in the internal sagittal stratum. The external (inferior) sagittal

stratum was held to be a temporooccipital association bundle. However, the evidence that the superior longitudinal bundle is identical with the optic radiation is overwhelming. This has been summarized recently by Putnam in his comparative study of the geniculostriate visual system of mammals, as follows:

1. In children and young animals, fibers may be traced from the inferior longitudinal fasciculus into the external geniculate body anteriorly, into the calcarine cortex posteriorly and into no other portion of the brain. The internal sagittal layer becomes myelinated at a much later period.

2. In appropriate cases, in which there is a large lesion in the vicinity of the corpus geniculatum, but the visual system is left intact, the stratum sagittale internum degenerates, but the inferior longitudinal fasciculus persists unaffected, and can be followed from corpus geniculatum to cortex.

3. Small lesions of the calcarine cortex or of the inferior longitudinal fasciculus produce degenerations which can be traced in this tract into the corpus geniculatum, but which disappear from the stratum sagittale internum as it is followed forward.

4. In animals, the coarse fibers of the inferior longitudinal bundle can be traced into the corpus geniculatum, in which practically no fine fibers such as make up the stratum sagittale internum can be seen. In certain animals (dolphin, seal), the entire longitudinal fasciculus can be seen to arise in the corpus geniculatum, and nowhere else. In other animals (apes), the fibers can be traced into the calcarine cortex, and nowhere else.

5. Following localized lesions of the corpus geniculatum, secondary atrophy is more complete in the fasciculus longitudinalis inferior than in the stratum sagittale internum. This is reported to be true also after long standing blindness, but evidence is conflicting on this point.

To these direct arguments may be added an indirect one:

6. The inferior longitudinal fasciculus has a characteristic histology, which is like that of the optic tract, the great motor and sensory radiations and the acoustic radiation. No proved association system contains such coarse fibers, but we should expect to find them in an important projection system.

Poljak[19], in a recent extensive study of the association, callosal and projection fibers of the cerebral cortex of the cat, states that the afferent optic fibers run from the lateral geniculate body in the lateral sagittal layer of the occipitoparietal lobe during the greater part of the course as a well defined and isolated fiber system. They are of larger caliber than the efferent fibers of the

occipitoparietal cortical regions, viz: callosal, association and other fibers. Putnam[14] has summarized also the clinicopathologic data which support the same proposition. The evidence seems conclusive that the geniculostriate radiation is identical with the fasciculus longitudinalis inferior.

The Macula and Retinal Quadrant Fibers in the Optic Radiation.

The problem of the organization of peripheral quadrants and macula in the optic radiation and upon the cortex has received much consideration and investigation. But its solution was not possible until the plan of the organization of the geniculate body was revealed. Holmes[20] had postulated an anatomic interval between the fibers of the upper and lower quadrants in the optic radiation on account of the prevalence in occipital lobe lesions of quadrant defects bounded by horizontal lines. Rönne[21] accepted this view and suggested that the macular fibers occupy this interval.

Putnam[14] has assembled the known facts of research in this field which, added to his own, shows the following to be true: (1) Retrograde degeneration from lesions in the upper striate area extends along the upper third of the optic radiation (inferior longitudinal fasciculus) to the mesial horn of the dorsal genicular nucleus (upper retinal quadrant area). (2) Degeneration from lesions in the lower calcarine area extends along the lower third of the optic radiation to the lateral horn of the geniculate nucleus (lower retinal quadrant area). (3) Degeneration from lesions of the posterior striate area extends thruout the middle third of the optic radiation to the large dorsal geniculate area (macula reception area). There was some fiber overlap from the middle region into the upper and lower edges of the radiation.

Organization of Retinal Quadrants and Fovea in the Visual Pathway.

When we trace degeneration from the upper and lower and posterior calcarine areas respectively to the medial, lateral and dorsal part of the lateral geniculate, the sites where the upper, lower and central retinal areas have already been found to be projected in the order named, we are in a position to follow the plan of organization from retina to cortex. This may be stated as follows:

The upper outer retinal quadrant is projected in the upper outer quarter of the optic nerve, the upper part of the chiasma,

the mediodorsal part of the optic tract, the medial horn of the dorsal nucleus of the lateral geniculate, the upper third of the optic radiation to the dorsal (upper) part of the area striata.

The lower outer retinal quadrant projection occupies the corresponding portion in the nerve, the lower part of the chiasma, the medioventral part of the tract, the lateral horn of the geniculate nucleus, the lower third of the radiation to the ventral (lower) part of the area striata.

The central retinal area is projected at first laterally and then axially in the optic nerve, intermediately in the chiasm, laterally and intermediately in the tract, dorsally and extensively in the lateral geniculate, thru the middle third of the optic radiation to a large area in the dorsal region of the area striata.

The upper and lower nasal quadrant projection occupies corresponding portions in the nerve and chiasm, the upper above, and the lower below, and then approach homologous relationship with the temporal elements as already described. The medial foveal fibers cross intermediately in the chiasm to seek homologous relationship also with the temporal foveal fibers of the opposite side.

It may be added that the hypothesis once proposed by Wilbrand[22] that there is a bifurcation of the macula fibers in the chiasm has not been supported by histologic studies. Moreover, the fact that the hemianopsia associated with the total interruption of the optic tract extends to the fixation point, takes away the physiologic support of that proposition.

There is in the neurologic literature apparently no complete report of isolated lesions limited to the lateral geniculate. The newer structural knowledge of the visual pathway, as formulated by Brouwer and Zeeman, Rönne, Putnam and others, should lead to physiologic and diagnostic advance, since it will put workers in this field on the watch for corresponding localizing data and furnish pathologists with morphologic criteria for the interpretation of selective structural lesions. It will be interesting to watch the course of neurologic and ophthalmologic development in relation to the new anatomic concepts.

Granting the validity of the visual anatomy formulated as here recounted, it may be justified to postulate types of neurologic and ophthalmologic visual defects which would logically follow lesions that selectively destroy the different parts of the complexly organized visual projection systems that have been revealed by modern research.

For example, from the viewpoint of the scheme of localization above described, it is to be expected that a small lesion de-

stroying the mesial horn of one lateral geniculate should give homonymous upper retinal quadrant defects on the same side, and, on account of image inversion in the eye, a corresponding deficiency in the opposite lower fields. A total lesion, say, of the mesial limb of the left geniculate should determine a right homonymous lower quadrantopsia. A destructive lesion similarly located in the right lateral geniculate would, of course, determine a left lower homonymous quadrantopsia. This should follow from the discharge of the upper retinal quadrants into the mesial limb of the lateral geniculate.

We should expect a destructive lesion in the lateral limb of the left geniculate, on the contrary, to produce a right upper homonymous quadrantopsia; while a lesion similarly located on the right side should produce corresponding field defects in the left upper quadrants, the reason being the connection of the lower homonymous retinal quadrants with the lateral limb of the lateral geniculate.

A lesion restricted to the dorsal part of the geniculate, which serves as a reception station for macular fibers, should produce homonymous central hemianopic scotoma, which might be further recognized as upper or lower, depending on whether the location was lateral or mesial in the genicular macular area, since the upper macular area lies medial to the lower in the dorsal part of the geniculate, as already stated.

A lesion limited to the ventral region of the geniculate should give monocular field defects in the "peripheral lune," or temporal crescent, of the visual field, which is imaged unilaterally upon the ventral edge of the nasal retina and is subject only to monocular vision. Disturbances of monocular vision have been studied by Behr and Lutz, but, as stated above, the anatomic phases of the representation of monocular vision in the optic radiation and cortex is not yet understood.

Lesions localized in the optic tract should result in limitation of vision definitely related to the plan of organization of its fibers, as described above. Visual defects in the case of lesions limited to the tracts have been described, but not from the viewpoint of the newer anatomic concepts.

With a primary or secondary involvement of the superomesial tract area, we should expect to find a visual defect similar to that resulting from a destructive lesion in the mesial limb of the dorsal nucleus of the lateral geniculate, (i. e. an opposite lower homonymous quadrantopsia) since this fiber area of the tract projects to the geniculate region of the lateral geniculate.

A similar lesion of the inferomesial tract fibers should give a picture resembling that which should follow a destruction of the lateral geniculate horn, i. e. an opposite upper homonymous quadrantopsia, since the lower retinal quadrant fibers arborize in the lateral geniculate horn.

A lesion interrupting the optic tract from the lateral border should be evidenced preponderantly in homonymous macular hemianoptic scotoma, because the macular fibers would tend to be interrupted in advance of those of the peripheral retinal areas, as may be seen by a study of Figure 3.

We should not expect such homonymous defects in tract lesions to be strictly congruous and superimposable, because clinical experience has shown, as Traquair[23] emphasizes, that scotomata due to tract damage tend to be incongruous, the homologous fibers from the two retinae apparently not yet having come into exact apposition. To what extent homonymous scotomata resulting from lesions of the lateral geniculate shall be found to be congruous, is an interesting question.

The character of the lesions limited to the genicular region may, in the present state of our knowledge, be formulated only in hypothetic terms. The problem of localization from limited damage in the optic radiation, however, may be stated in the more tangible terms of considerable clinical and investigative experience.

Regarding lesions in the anterior portion of the radiation, as will be noted later, quadrant defects are produced for color and for form and colors.

More posteriorly in the optic radiation (stratum sagittale externum), lesions interrupting its upper edge produce opposite lower homonymous quadrantopsia. This was shown by Lenz[24] who reports two cases at different times exhibiting interruption of the upper radiation. This is to be expected, since the upper retinal quadrants commanding the opposite lower fields project, as we have seen, via the medial horn of the geniculate thru the upper edge of the radiation to the upper striate cortex.

Brouwer[25] describes a case in which all the area striata was degenerated excepting a small area of the upper calcarine lip at the level of the posterior horn of the lateral ventricle. The anteromedial part of the geniculate was spared and the remaining portions of it degenerated. Winkler[26] reports three cases of lesions of the upper optic radiation and atrophy of the mesial limb of the lateral geniculate. Others giving similar reports are von Stauffenberg[27], Neissl von Mayendorf[28] and Archambault[29].

As regards the effect of a lesion in the middle or macular zone of the optic radiation, Brouwer[25] showed that such a lesion at the tip of the occipital lobe involving the cortex and the posterior end of the radiation gives rise to atrophy in the dorsal (macular) part of the lateral geniculate. The field defect resulting from such a lesion is central.

As for lesions in the lower radiation, Putnam's reconstructions show that the resultant degeneration in this location extends to the lateral geniculate, in the region of its lateral horn (site of lower retinal reception). The fields were not given in this case, but scotomata caused by the lesions should lie in the opposite upper homonymous quadrants. Other workers who show that damage to the lower part of the radiation causes atrophy of the lateral limb of the geniculate are Neissl von Mayendorf[28], Pfeifer[15], and Winkler[26].

Clinical records, as by Holmes[29] and others, support the "vertical" organization in cortex and optic radiation as given here. Lesions involving the occipital pole, and thus damaging the posterior striate, gave rise invariably to central visual defects. Experience in the Great War showed that damage to the upper lip of the area striata gave rise to lower quadrant defects. The cases revealing lower calcarine lesions generally failed to survive on account of the proximity of the large venous sinuses and the vital centers.

The above suggestions, in part hypothetic, regarding visual deficiencies following selective destruction of the organized systems in the visual pathway, as formulated by the Amsterdam workers, are presented here only as a matter of interest and as a means of stimulating anatomic, clinical and pathologic observation and research.

The common statement that supragenicular lesions in the visual pathway produce homonymous scotomata that are congruous and superimposable needs to be qualified. Holmes and others have shown that congruity characterizes such homonymous scotomata when produced by lesions in the cortex and posterior part of the optic radiation. Congruity, however, is less evident in the case of lesions of the anterior part of the optic radiation. This is clearly seen, especially in the work to be noted later of Cushing and Lillie, who have shown, that in such lesions as temporosphenoidal tumor or abscess, incongruity of resulting homonymous scotomata is common. Minkowski[30] arrived at the same idea from his studies.

Another interesting proposition, emerging from the data of Putnam's research and study, is that the macular area is not only

located at the occipital pole, as was earlier claimed, but that its cortical site is disproportionately large. This is significant as illustrating the principle of relatively larger cortical representation for the more highly specialized functions. The large size of cortical representation coincides with the relatively large proportion of macular elements thruout the length of the visual pathway. Moreover, it has been shown by Lenz[24] and Cobb[31] that the posterior part of the visual cortex is cytologically richer and more highly complex.

The Shape and Size of the Area Striata.

The form, size and cytoarchitecture of the area striata have been known for a long time from the studies of Brodmann[32], Campbell[33], and others. Its relationship to visual function was established by such work as the myelinization and degeneration studies of Flechsig, by the exhaustive studies of cases of peripheral blindness in man, by Lenz, by the work of Minkowski on the localization of visual function in the cat, and by others. The area is round or oval in marsupials and lower rodents, and becomes more complicated in higher animal forms. In the ape, it occupies the mesial and lateral surfaces. The area shows a tendency, most striking in primates, to become depressed in the surface of sulci and fissures, notably the calcarine, leaving its boundaries upon the surface. In man, the area striata not infrequently extends around the occipital pole upon the lateral surface, and may then become folded in the line of its axis to form a sulcus calcarinus lateralis.

The size of the area striata was determined in five normal cases by Putnam (IV). The smallest area was 18.3 sq. cm., the largest, 24.57 sq. cm., the average, 20.7 sq. cm. He finds the size of the area somewhat smaller than that given by Elliot Smith[34]. According to the latter, it varies from 2700 to 4000 sq. mm. averaging 3000. The cortical projection area is thus small in comparison with the total cerebral surface. Brodmann[32] emphasized the fact that there are marked individual variations of the striate area as regards size, position, form and relations to the sulci. There are variations as to symmetry and lateralization, which in some cases are marked. Brodmann suggested that these variations may in part explain the discrepancies between surgical and perimetric findings, as well as the divergencies of view represented by the von Monakow-Henschen schools of controversy as to diffuse versus circumscribed localization. The calcarine area is subject to "vagaries" and is an unreliable landmark for orientation (Putnam).

Histologically, the area striata is richer and more complex in its posterior part than anteriorly, as was shown qualitatively by Lenz and quantitively by Cobb[31].

It is also of interest, that there is a similar increase in the central retinal regions as compared with those in the peripheral quadrants. Zwanenburg found about 65 per cent of the retinal projection neuron bodies located 25 degrees from the fovea.

It would seem that the size of the "fovea" may be less restricted than is generally assumed in clinical and physiologic thinking. This is suggested by the reorganization of retinal function that frequently occurs after homonymous hemianopsia. The half visual fields are reorganized spontaneously on the normal plan, a "pseudofovea" develops and vision is as before, the subject being unaware of his hemianopsia. (Fuchs[35].)

The Older Views as to the Localization of the Retinal Quadrants and the Macula Upon the Cerebral Cortex.

The experimental data on the problem has been reviewed by Minkowski and Brouwer and Zeeman[36] ('01). A great deal of war and prewar work was devoted to an attempt to solve the problem: In consequence of their observations, many workers, such as Pierre Marie and Chatelin in France, Axenfeld in Germany, Gordon Holmes and Lister in England, and others, were able to develop certain working hypotheses as to the cerebral localization of visual impressions. Out of such studies, there emerged definitely the view that the visual area is located along the calcarine fissure; the upper quadrant above the calcarine, the lower below, and the macula upon the cortex at the occipital pole. The best statement, based on the clinicopathologic method, of the organization of the area striata has been presented by Holmes[20] ('18), and is here given for comparison with the results obtained by the anatomic methods. Holmes had not the opportunity of exact histologic study of material, and was unable to generalize except by inference upon the organization of the geniculate body and optic radiation. However, in general outlines, the visual cortical localization as he formulated it agrees with the conclusions arrived at by the anatomic study and reconstruction of experimental embryologic and pathologic material.

As a result of his study of cerebral lesions during the war, he formulated the following views, viz.:

1. That the upper half of each retina is represented in the dorsal, and the lower in the ventral part of each visual area.

2. That the center for macular vision lies in the most posterior part of the visual cortex, probably on the margins and on

the lateral surfaces of the occipital poles. The macula has not a bilateral representation.

3. That the center for vision subserved by the periphery of the retina is located in the anterior portions of the visual areas, and the serial concentric zones of the retinae from the macula to the periphery are probably represented in this order from behind forwards in the visual cortex.

4. That those portions of the retinae adjoining their vertical axis are probably represented in the dorsal and ventral margins of the visual areas, while the retina in the neighborhood of the horizontal axis is projected onto the walls and floor of the calcarine fissures.

5. That severe lesions of the visual cortex produce complete blindness in the corresponding portions of the visual fields, or if incomplete, an amblyopia, color vision being generally lost and white objects appearing indistinct, or only more potent stimuli, such as abruptly moving objects, may excite sensations.

6. That the defects of vision in the fields of the two eyes are always congruous and superimposable, provided that no abnormality of the peripheral visual apparatus exists.

Holmes[20] had not the opportunity of histologic study of his material, and was unable therefore to trace degenerative changes in the optic radiation and lateral geniculate. The views he formulates upon cortical localization have been verified, clarified and extended by the more accurate anatomic methods of study.

The newer knowledge of arrangement and organization in the visual pathway holds a measure of validity hitherto missing, since it is based on the anatomic method and lacks the degree of uncertainty that in the older methods led to different schools of interpretation of similar data. Further study along this line should continue to clarify our anatomic knowledge and to lead to the development of clearer physiologic and clinical concepts in this difficult field.

There have been recent diagnostic advances in cerebral tumor localization which have been expressed in the form of syndromes, which are definitely related to the organization of the fibers of the visual pathway. One such syndrome, based entirely upon ophthalmologic data, forms the clue to the localization of a tumor in the inferior surface of the frontal lobe.

The Basal Frontal Syndrome.

It has long been known that the fibers of central vision tend to occupy an axial position in the optic nerve. There is a syn-

drome associated with the functional interruption of this highly specialized maculopapillar bundle which is still, I believe, not generally sufficiently recognized. Since there has been some recent advance in our knowledge of this syndrome of the basal frontal tumor, I shall include a brief account of it in this paper.

In 1906, Wilbrand and Saenger[37] reported several cases of tumor of the frontal lobe which exhibited unilateral choked disc. Six years later, they reported unilateral choked disc from a basal frontal lesion, and mentioned having seen similar cases. No perimetric studies were made, however, and there was, therefore, no note taken of the presence or absence of homolateral central visual defect.

In 1911, Kennedy[38] reported six cases of basal frontal lobe tumor. His report contained an observation of great diagnostic importance, namely, that a tumor in this site produces a homolateral central scotoma. Some of his cases showed a contralateral choked disc. Five years later, he reported a case of aneurism in the same region, producing a similar picture.

Recently Lillie[39], of the Mayo Clinic, has given a report upon a series of thirteen cases which clarifies this syndrome. The following is an abbreviation of a case history of the series represented by him which illustrates the indispensability of ophthalmologic data for localization of basal frontal tumor.

A girl, 15, came to the Mayo Clinic September 1, 1926, because of nervousness and headache. Symptoms of five years duration included poor memory, and behavior changes, and inability to go beyond the fourth grade in school. For three years, attacks of sleep occurred, at times lasting for four or five days. Also, she had periods of ataxia lasting four to ten days. During the last two and a half years, she had suffered five attacks of generalized convulsions. During the last two months, severe right frontal headache kept her awake at night, and vision began to fail.

Negative results were obtained in physical, neurologic and serologic examinations, and roentgenologic examination indicated a slight increase of intracranial pressure.

Ophthalmologic examination showed she had normal vision in the left eye (6/6), and could only count fingers with the right eye.

Pupillary reflexes, ocular movements and fundi were normal. The right pupil was larger than the left.

The perimetric fields showed right cecocentral scotoma, with form and color fields normal. The left field was normal for form and color.

Surgical exploration showed a right basal frontal lobe tumor, which was not removed. Vision returned to 6/5 R, and 6/5 L. The reflexes, ocular movements and fundi remained normal, and no scotoma could be demonstrated in the right eye.

The disc did not choke, probably on account of the slow growth of the tumor. The cecocentral scotoma with otherwise normal vision gave the clue to the location of the tumor.

In this series, the characteristic feature was a central or cecocentral scotoma on the side of the lesion when it was unilateral, and similar bilateral central visual defect when the lesion extended across the midline so as to implicate also the opposite optic nerve (3 cases). Continued pressure enlarges the cecocentral scotoma to include the peripheral field, until only a small isle of vision remains, medial or lateral to the fixation point (3 cases). Persistent pressure produces complete amaurosis on the side of the lesion (1 case). There was one case of bilateral complete amaurosis, due to an unusual pushing of the temporosphenoidal lobe against the chiasma.

The remaining cases presented homolateral central or cecocentral scotoma. Lillie emphasizes that papilledema is not of important localizing value. This may be seen in the summary of his series included in his paper. His series contain cases of the bilateral presence and bilateral absence of choke, and of unilateral choke both on the side of the lesion and on the opposite side. He says: "The fundal changes are not so characteristic. In seven cases, there were bilateral choked discs, while in only four was there a normal or pale disc on the side of the lesion with an associated choked disc on the opposite side. In the other three cases, the condition of the fundi varied from bilateral pallor of the discs with some blurring, to a slight blurring of one disc and a definite choked disc on the opposite side. Apparently there is no definite sequence in the development of the choked disc or pale disc, as a few cases were examined when the fundi were absolutely normal, and a few days to a week later an early choked disc was found, either beginning on the side of the lesion before the opposite side was affected, or just the reverse. Again, the normal disc had become pale, without evidence of edema of the disc developing on the side of the lesion. Nine of the fourteen cases showed evidence of bilateral edema of the disc during the period of observation, which would suggest that a retrobulbar picture, with a concomitant choked disc, is not the usual condition.

The view, that there should be of necessity a contralateral choked disc, with the unilateral disc remaining normal, is theor-

retically plausible, but it does not occur with sufficient frequency to constitute a dependable localizing feature.

It is also interesting, that in this series the "loss of the sense of smell occurred too rarely to be of localizing value."

The development and clarification of the basal frontal syndrome is very important. Frontal lobe tumors are not infrequent, and a substantial number of them (15 per cent in this series) are basally located. It is true that postfrontal tumor is generally the easiest of all to localize, since, on account of their proximity to the great cortical motor centers, they cause irritative or paralytic phenomena of unmistakable localizing value. Moreover, a tumor in this location tends to disturb, when on the left side, the motor speech center.

But localization of frontal tumor more anteriorly situated may be difficult or even impossible by other than ophthalmologic methods. Physical, neurologic, and roentgenologic examination in this series generally yielded data inadequate for localization.

Lillie makes the important point that chiasmal lesions can produce scotomatous field defects similar to the basal frontal tumors, but these are rarely associated with choked discs.

When the general evidence indicates the presence of intracranial neoplasm and localizing data is not evident, the problem has tragic possibilities for the patient. When, however, examination discloses the ophthalmologic syndrome noted above, the seat of mischief may be placed in the basal frontal region, as clearly shown in the Mayo Clinic series.

Lillie's summary outlines his conclusions in brief compass as follows:

1. Basal lesions of the frontal lobe can be localized accurately from the ophthalmologic examination.

2. In a unilateral lesion, homolateral central or cecocentral scotoma, associated with either a normal, pale, atrophic or choked disc, with contralateral normal central vision and choked disc, is characteristic.

3. In a bilateral lesion, bilateral central or cecocentral scotomas are present, associated with bilateral choked discs.

4. Basal lesions of the frontal lobe are common (15 per cent), and can be diagnosed as readily and as accurately ophthalmologically as chiasmal lesions.

The Syndrome of the "Temporospheroidal" Lobe Tumor.

Tumor localization in the temporal lobe has been considered until fairly recently to offer special difficulties. Up to a few years

ago, this territory was called the "silent area" because of its assumed "physiologic latency." It was held that tumors here are totally devoid of focal symptoms and produce symptoms only by pressure on neighboring parts. However, thru the work of Kennedy, Cushing[40] and later of Lillie[41] and others, an ophthalmologic syndrome has been devolped which is highly diagnostic of tumor or its equivalent in the region of the brain overlying the "temporal knee" of the optic radiation. A brief account of this ophthalmologic method of intracranial localization will be here outlined.

Kennedy,[42] in 1911, gave the symptomatology of temporosphenoidal lobe tumor, but omitted the field defects which were later established as quantitative perimetry became developed. Cushing,[40] in 1921, published a classic contribution on the subject, which supplied the ophthalmologic feature omitted by Kennedy, and established a syndrome for the localization of a tumor in this region. More recently, Lillie, of the Mayo Clinic, has published a large series of cases, and his findings have thrown much light upon the localization of tumor in the temporal region.

One of Cushing's[40] case reports, which I have taken the liberty to abbreviate, follows:

Dorothy D., aged 19, developed general signs and symptoms of brain tumor. Among these were headache, fainting and convulsions. The tumor was not localizable by general examination, which showed her in excellent physical condition, nor by neurologic examination, which showed only slight left hypesthesia, without motor involvement, tho deep reflexes were possibly more brisk on the right.

Ophthalmologic examination revealed bilateral choked discs (R., 1.5; L., 3 D). A chart of the perimetric fields showed left homonymous defect. The bilateral choked discs and the homonymous quadrant defects gave the clue to the temporal location of the tumor, which proved at operation to be a gliomatous cyst.

Cushing analyzes many cases of temporal lobe tumor in detail. The symptomatology, aside from the features resulting from general pressure which are more or less common to all intracranial tumors, such as headache, papilledema, vomiting, convulsions, mental alterations, etc., was complicated by varied effects of special pressure upon neighborhood parts, as upon pathways projecting to the peripheral tissues and to the cerebellum, etc. In thirteen of his series of fifty-nine cases, there were uncinate attacks, which of course has, as Hughlings Jackson showed, great

localizing value. In some others, there were persistent, highly organized visual hallucinations projected to the defective visual fields.

The most valuable localizing data, however, were obtained by the use of the ophthalmoscope and the perimeter, Quantitative perimetric studies were accurately made by Walker,[43] who has done so much to develop this field. Small graded test objects were employed, which generally revealed the presence of homonymous field defects, the latter ranging from slight quadrant flattening to complete hemianopsia. Choked disc was also frequently found. The quadrant defects were due to impingment upon the geniculostriate fibers of the superjacent tumor. The total hemianopsia present in some advanced cases was caused by the pressure disturbance extending to the optic tract. Cushing's paper should be read by all who are interested in this subject.

In Lillie's series of one hundred and sixty-eight cases of certified cerebral tumors examined in the Mayo Clinic during the previous five years, sixty of them were located in the temporal lobe. The location was proved in all but three by operation, and in these three, it was proved by necropsy.

In this series, the association of the occurrence of the two ophthalmologic features—quadrant changes for colors, or for form and colors, and choked disc—occurred together frequently, as in Cushing's series.

Disc Changes: In forty-five of his sixty cases, there was bilateral choke ranging from ½ to 7 diopters. The swelling was as frequent on the opposite side as on the same side. It was equal in twenty-four cases. One case showed a definite bilateral secondary atrophy without edema. There were normal fundi in thirteen cases, and in one, mild pallor of both discs. The blind spots were enlarged excepting in the cases without edema, and in one with mild disc pallor.

Perimetric Field Changes: The author classified his fields as (1) normal, (2) homonymous quadrant defects for colors and for form and colors, and (3) homonymous hemianopsia for colors and form and colors.

In regard to changes in the perimetric field he showed:

1. That the perimetric fields were normal for colors and for form in eight cases, but in three of these, the blind spots were enlarged owing to the papilledema.

2. That ten cases showed quadrant defects for one or more colors, in the upper quadrant in six cases, and in the lower quadrant in four.

3. That the cases with normal form field showed homonymous hemianopsia for colors; a complete homonymous hemianopsia for form and colors was present in fifteen cases.

The perimetric fields could not be charted in three cases because of mental impairment in the patient. Eleven cases showed definite aphasia.

In seventeen of these sixty cases, localization was impossible from neurologic findings, which were only of general character. On the other hand, fifteen cases, definitely localized by the neurologic findings, gave ophthalmologic data of no localizing value. In six of the latter, the fields were normal, and in nine, mental impairment precluded field charting.

Definite localization was possible in forty-three by ophthalmologic data alone, and in forty of the sixty cases, by neurologic findings alone. The fact that definite localization was possible oftener from ophthalmologic than from neurologic findings shows the great localizing value of this syndrome. Space does not permit the admission of detail in connection with this series.

The essential points, only, are given to emphasize the great localizing value of the combination of quadrant defects and choked discs in temporal lobe tumor. Lillie's cases justify his conclusions, which follows:

1. That temporal lobe tumors are common (sixty out of one hundred and sixty-eight verified cerebral tumors).

2. That homonymous quadrant defects, either for colors or for form, associated with bilateral choked discs, are characteristic of this location (forty-three out of fifty-one cases).

3. That the choking of the disc was higher on the contralateral side as often as on the homolateral side.

4. That visual hallucinations were absent.

5. That an ocular syndrome could be established for early temporal lobe tumors; namely, normal visual acuity, bilateral choked discs, and homonymous quadrant defects either for colors or form.

This brief and general statement upon these series emphasizes the relationship between the physiologic and anatomic disorganization of certain parts of the visual pathway, and the special symptomatologic feature which constitute localizing syndromes.

In the basal frontal syndrome, the special diagnostic feature results from the physiologic delicacy of the maculopapillar bundle, which is thrown out of function by space restricting growths pressing upon it and producing central visual defect. In

the temporal lobe syndrome, one of the two important ophthalmologic diagnostic features—the quadrant defects—is also due to local pressure disturbane upon the geniculostriate fibers, arched widely forward before sweeping posteriorly to their termination in the striate cortex. Function frequently returns when the pressure is removed, showing that functional derangement may be marked and of long duration before atrophy or degeneration supervenes.

Summary and Conclusions.

1. The efficiency of the method of localization of intracranial lesions by a study of modification or defect of the neural projection systems, depends upon definite anatomic and physiologic knowledge of these systems.

2. Advance in such knowledge is necessary as a means of reduction of the fraction of intracranial lesions still unlocalizable by our present methods.

3. Definite knowledge of the organization and arrangement of the elements of the visual system has been until recently unsatisfactory. Recent advances in the anatomy of this system, as developed by the basic anatomic researches of Brouwer and Zeeman, Putnam and others (upon normal and pathologic material), is here recounted and summarized.

4. Researches by these methods have shown that there is a more or less circumscribed localization of the elements for peripheral and central vision in the optic nerves, chiasm, tracts, lateral geniculate bodies, optic radiations, and striate cortex.

5. The symptomatologic picture that should result from lesions localized in different parts of the visual system, as thus organized, is discussed.

6. A brief account is given of some recent valuable gains in our knowledge of localization of basal frontal, and temporal lobe tumors thru ophthalmologic findings which result in part from functional impairment of certain parts of the visual projection pathways.

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DISCUSSION.

DR. JAMES F. MCDONALD, Omaha, Neb., (closing): As regards the cortical representation of the macula, the question is still under discussion whether it is bilaterally represented. The problem is still in the theoretic, and therefore controversial, stage. The solution of the problem of the projection system which resisted for so long the clinicopothologic method of research has been localized with considerable accuracy in the optic nerves, chiasma, tracts, radiation and cortex by the application of anatomic research methods. No suprachiasmal cross connections of the visual pathways has been anatomically definitely demonstrated, distally to the cortex. A definite connection of the areae striatae has been recently demonstrated by Poljak, (cat), tho the physiologic significance of this connection has yet to be demonstrated. This point has been summarized recently by Putnam, and Kluver, who gave an exhaustive bibliography on that point.

As yet we have not definite data for selection among the various theories offered in explanation of macular escape which may be the nearest to the truth.

Macular escape, so common in vascular lesions, is held to be due to the dual blood supply of the occipital pole. Some hold that the functional superiority of the cortical macula holds it above the functional threshold, when the peripheral fields fall below. Others hold that the large extension of the cortical macula in its protected medial location accounts for its escape. The older theories of bilateral representation of Wilbrand, Heine and Lenz are also still in the field, tho the site of fiber bifurcation is no longer held to be the chiasm.

Men like Holmes and Traquair in clinical neurology and ophthalmology hold that the macula is not bilaterally represented. It appears that the solution of this complicated question, like that of the organization of the visual pathway, may not be solved by the clinicopathologic method, but like the latter may yield to a change in the manner of approach.

NEW VESTIBULAR COMPLEXES FOR LOCALIZATION OF BRAIN LESIONS.

(An Analysis of 139 Verified Brain Lesions.*)

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In 1926, we published from this Clinic (Fisher and Grant, Journal für Psychologie and Neurologie, Band 34, Heft 3 u. 4) our experience with the vestibular reactions in 116 verified intracranial lesions. In the present contribution, we propose to review the vestibular reactions especially in pretentorial lesions, with a view to determining whether they present syndromes which would be an aid to localization.**

This series of cases by no means represents the total number which have passed thru the clinic and were studied by the writers. Several thousand patients were examined, but unfortunately many were unverified, while others, tho verified by autopsy or operation, could not be utilized because the charts were incomplete.

The following table shows the number of lesions constituting this series with their location in the respective regions of the brain.

A. Supratentorial.

I. Hemispheric.

1.	Frontal Lobe	11
2.	Frontoparietal	2
3.	Frontotemporal	1
4.	Motor Area	10
5.	Parietal	1
6.	Temporal	6
7.	Motor-Temporal	2

*From the Neuro-Surgical Clinic of Dr. Chas. H. Frazier, University of Pennsylvania, and the Neuro-Otologic Clinic of the University of Pennsylvania.

**This study was made possible thru the cooperation and interest of Dr. Charles H. Frazier, whose Neuro-Surgical Clinic supplied most of the cases, and Drs. W. G. Spiller and Theo. H. Weisenburg, whose encouragement and aid helped in the development of neurootology.

8.	Occipital	3
9.	Junction Temporoparietooccipital	4
10.	Intraventricular	2
11.	Corpus Callosum	1

II. Midcentral Tumors.

1.	Pituitary	11
2.	Suprasellar	6
3.	Third Ventricle	3
4.	Corpora Quadrigemina	1
5.	Pineal	1
		65

B. Subtentorial.

III. Cerebellar Tumors.

1.	Midline	6
2.	Midline, with pontile involvement	2
3.	Cerebellar lobe	25
4.	Cerebellar lobe with involvement of angle	4
		74

IV.

1.	Cerebellopontile angle tumors	37

For the purpose of more clearly presenting the findings necessary for the diagnosis of a brain lesion by means of the vestibular tests, this paper has been divided into the following sections:

1. Findings Indicative of a Brain Lesion.
2. The Value of Vestibular Tests in the Determination of Increased Intracranial Pressure.
3. Findings Indicative of a Supratentorial and Subtentorial Lesion.
4. Findings Indicative of the Laterality of the Lesion.
5. Vestibular Group Findings Diagnostic for Each Anatomic Area.
6. Differential Diagnostic Signs Between Various Anatomic Areas.

Findings Indicative of a Brain Lesion.

1. Spontaneous vertical nystagmus: Small nystagmoid movements of the eyes on looking to the extreme right and left may be present in normal individuals, but a nystagmoid movement of considerable amplitude in any direction is pathologic.

These spontaneous nystagmoid movements may be horizontal—to right or left, rotary—to right or left, vertical—up or down. While it is true that well developed spontaneous nystagmus in any direction indicates pathology, it is only the vertical spontaneous nystagmus which is of interest in this study, because such a nystagmus always indicates a brain lesion. The other types of spontaneous nystagmus, horizontal or rotary, may be produced either by a brain or peripheral lesion (labyrinthitis, etc.) and therefore are not always indicative of a brain lesion. Vertical nystagmus, on the other hand, is never observed in peripheral lesions, and is therefore a true infallible and pathognomonic sign of brain pathology. This observation was first made by Robert Barany and is fully corroborated in our experience.

2. Poor pelvic girdle movements: This test is performed in the following manner. The patient stands erect, with heels and toes together, eyes closed, and arms to the side. The examiner then attempts to push the patient to the right, to the left, forward and backward. The ability of the pelvic girdle to compensate for the various displacements from the upright position is then noted. A normal individual will compensate against this attempt to overthrow by flexing his body at the hips, which prevents loss of balance. An individual with a brain lesion loses this flexibility of the pelvic girdle, and when an attempt is made to overthrow him, he falls over more readily.

3. Marked disproportion between nystagmus and vertigo after turning.

4. Marked disproportion in the activity or duration of the responses from the horizontal and vertical semicircular canals of the same side after douching, such as:

a. No nystagmus from the vertical canals, but good nystagmus from the horizontal canal.

b. Much delayed, or slow nystagmus from the vertical canals, and good, active, rapid responses from the horizontal canal.

5. Loss of nystagmus from the vertical canals, but past pointing present.

6. Perverted nystagmus from either horizontal or vertical canals after douching. The perversion of the nystagmus from the horizontal canal may vary from a slight obliquity to almost a vertical nystagmus, or it may be rotary instead of horizontal. The more marked the perversion, the stronger the evidence that the lesion is intracranial.

7. Vertigo, or past pointing, in the wrong direction after stimulation.

8. Dissociated movement of the two eyes, after stimulation.

9. Loss of all vestibular responses after stimulation, with good hearing.

10. Conjugate deviation of eyes after stimulation, instead of a full nystagmus.

In all cases presented, one or more of the above findings existed. While the presence of these phenomena indicate a brain lesion, they do not indicate the nature of the disturbance. The lesion may be a tumor, large or small, a degenerative process, inflammation, hemorrhage or even a toxic state.

The Value of Vestibular Tests in the Determination of Increased Intracranial Pressure.

Up to the present analysis, it was considered that some of the abnormal responses to ear stimulation, such as loss of vertical canals' function, perverted nystagmus from the horizontal canal, and spontaneous vertical nystagmus, were indicative of an increase in generalized intracranial pressure. However, in this analysis, it was found that in many cases with the spinal manometric pressure tremendously increased, none of the above named phenomena were present: and conversely, in some of the cases where these same vestibular phenomena were marked, the spinal manometric pressure failed to show any increase. It is obvious, therefore, that these vestibular findings do not indicate generalized increased intracranial pressure, as such. These abnormal findings, however, indicate a brain lesion, and since they are not always produced by actual destruction, they must be produced by pressure of some sort, probably local pressure.

Findings Indicative of a Supratentorial and Subtentorial Lesion.

It is needless to dwell on the importance of differentiating between supratentorial and subtentorial lesions from a surgical standpoint. This analysis of one hundred and thirty-nine brain cases offers an excellent opportunity to determine the findings by which such a differentiation is possible.

One of the most salient diagnostic signs is the patient's susceptibility to ear stimulation. Patients with subtentorial lesions do not become nauseated, do not vomit or perspire, regardless of the amount of stimulation administered. On the other

hand, patients with supratentorial lesions are frequently quite susceptible, the degree of susceptibility varying with the location of the lesion.

It is extremely important to note that susceptibility of a patient to ear stimulation definitely excludes a posterior fossa lesion, but immunity from disturbance does not necessarily place the lesion subtentorially, because certain supratentorial lesion patients are also unsusceptible to ear stimulation. It is also well to remember, that normal individuals are frequently upset by ear stimulation. Therefore, this phenomenon during the examination of a patient does not necessarily indicate the presence of a brain lesion, but it does indicate, that if a lesion is present, it is not subtentorial. Should a patient show no susceptibility during a vestibular examination, we must rely upon some of the other findings for our differentiation, such as poor pelvic girdle movements, impaired vertigo and past pointing, presence of a marked spontaneous nystagmus, or evidence of actual involvement of the horizontal canal's fibers. These various findings, when taken in combination, enable us to make a differentiation.

In the cases analyzed, susceptibility was noted in lesions of the frontal lobe, motor area, the temporoparietal area, pituitary and suprasellar regions. Susceptibility to stimulation was absent in lesions of the temporal lobe, occipital and all subtentorial lesions.

Findings Indicative of the Laterality of the Lesion.

The side on which a cerebral hemispheric tumor is located may be indicated by the following findings:

1. Uncertain spontaneous pointing, or uncertain past pointing of the arm or leg after stimulation indicates lesion on the opposite side (seventeen cases). It is well to bear in mind that the mere ability of the patient to find the examiner's finger does not necessarily indicate normal pointing, but the manner in which the patient finds the examiner's finger should be carefully noted. Even if the movement is not definitely ataxic or asynergic it may be extremely uncertain and therefore pathologic.

2. Conjugate deviation after stimulation to the side of the lesion. (Seventeen cases.)

3. Interference with, or a total absence of all vestibular responses from one ear indicates a lesion of the opposite side. (Seven cases.)

4. Perversion of the nystagmus from the horizontal semicircular canal indicates a lesion on the same side. (Seven cases.)

By means of these signs the correct side was localized in thirty-one out of forty-three cases. The other twelve cases had no lateralizing signs.

The side on which a cerebellar lesion is located may be indicated by the following:

1. Uncertain pointing of the arm or leg on the same side. (Six cases.)
2. Interference with or loss of all vestibular responses after stimulation on the side of the lesion. (Nine cases.)
3. Impairment of nystagmus from the horizontal semicircular canal on the same side. (Eight cases.)
4. Interference with responses from the vertical semicircular canals on the same side. (Ten cases.)
5. Deafness on the side of the lesion.

In three cases the side of the lesion could not be placed, and in one case it was wrongly placed.

In cases of cerebellopontile angle lesions, the tumor is always found on the side on which the entire eighth nerve function is disturbed.

Vestibular Group—Findings Diagnostic For Each Anatomic Area.

Supratentorial.

Cererbal Hemispheric Lesions.

The following group findings are indicative of the areas described.

Frontal Lobe (eleven cases):

1. Definite susceptibility to ear stimulation (ten out of eleven.)
2. Definitely good past pointing after stimulation.
3. Absence of spontaneous vertical nystagmus.
4. Pelvic girdle movements good. (Poor in two only, but in those two the lesion extended towards the motor area.)
5. Nystagmus after turning normal or slightly exaggerated. (Never subnormal.)
6. Vertigo usually normal, or slightly subnormal, never exaggerated.
7. Vertical semicircular canal's function usually interfered with. (Seven out of eleven.)

8. Horizontal canal nystagmus usually not perverted. (Only three out of eleven had perversion.)

Motor Area (ten cases):

1. Susceptibility to stimulation slight. (Only three cases.)
2. Past pointing good in all cases.
3. No spontaneous vertical nystagmus.
4. Pelvic girdle movements poor in all.
5. Nystagmus after turning good—rarely slightly exaggerated.
6. Vertigo after stimulation definitely diminished in all.
7. Vertical semicircular canals function affected. (Nine out of ten.)
8. Horizontal canal nystagmus not perverted, only slightly so in two cases.

Temporal Lobe (six cases):

1. Susceptibility to stimulation absent.
2. Past pointing practically normal in all, except one.
3. Spontaneous nystagmus absent, except in one.
4. Pelvic girdle movements good in four, poor in two—probable motor involvement in the latter.
5. Nystagmus after turning normal, or exaggerated.
6. Vertigo after turning, diminished in four, exaggerated in two.
7. Vertical semicircular canals unaffected, except in two.
8. Horizontal canal nystagmus not perverted.

An unsual feature was noted in two cases. The duration of vertigo after stimulation was tremendously prolonged.

Occipital Lobe (three cases):

1. Susceptibility to stimulation absent.
2. Past pointing markedly exaggerated, both in extent and duration out of proportion to the vertigo.
3. No spontaneous nystagmus.
4. Pelvic girdle movements good.
5. Nystagmus after turning, normal.
6. Vertigo, after turning normal—not diminished.
7. Vertical semicircular canals unaffected.
8. Horizontal canal nystagmus perverted.

While it is realized that in all of the above cases the lesion did not actually limit itself entirely to the lobe designated, yet the greatest pathology was in the lobes considered. In some of the cases, however, the lesion definitely extended to more than one lobe and of those we have:

FRONTOPARIETAL (two cases):

The findings about the same, as in the frontal lobe, except that the pelvic girdle movements were poor in both of these cases.

JUNCTION-TEMPORO-PARIETO-OCCIPITAL (four cases):

1. Susceptibility to stimulation absent.
2. Past pointing uniformly good.
3. Spontaneous nystagmus absent.
4. Pelvic girdle movements good.
5. Nystagmus after turning, normal.
6. Vertigo normal, or slightly subnormal.
7. Vertical semicircular canals impaired.
8. Perversion of horizontal canals' responses absent.
9. All vestibular fibers (vertical and horizontal canals) on the side opposite the lesion show impairment or absence of function, with hearing on that side normal. As the lesion is located more posteriorly and becomes more definitely occipital, this characteristic becomes less evident in proportion.

MIDCENTRAL LESIONS.

PITUITARY (eleven cases):

1. Susceptibility to stimulation varies. There was none in five cases, but it was present to an extreme degree in six cases.
2. Spontaneous past pointing absent.
3. Spontaneous nystagmus absent, except very slightly in a few cases.
4. Pelvic girdle movements good.
5. Nystagmus after turning usually increased, altho in four cases was either normal, or slightly diminished.
6. Vertigo, generally speaking, normal or diminished. The number in which vertigo was definitely diminished was greater than those in which vertigo approached the normal.
7. Vertical semicircular canals' responses usually present.
8. Horizontal canal nystagmus not perverted.

SUPRASELLAR, INCLUDING LESIONS OF THE CORPORA QUADRIGEMINA, PINEAL BODY AND THIRD VENTRICLE (eleven cases):

1. Patients are usually somewhat susceptible to ear stimulation, and in cases where patients are clinically very sick, susceptibility is usually very marked.
2. Past pointing variable.
3. Spontaneous nystagmus absent.

4. Pelvic girdle movements poor.
5. Nystagmus after turning variable.
6. Vertigo and nystagmus not in proportion to each other.
7. Vertical semicircular canals, if interfered with at all, usually on one side only.
8. Horizontal canal nystagmus usually normal; in only three cases was it perverted.

When the lesion is very high, as in the region of the pineal, or corpora quadrigemina, there may be found evidence of involvement of the posterior longitudinal bundles, noted by dissociated movements of the eyes following ear stimulation.

Subtentorial.

For the purpose of analysis, we have divided these into cerebellar hemispheric lobe cases, midline tumors, cerebellopontile angle tumors and cerebellar tumors involving the angle secondarily.

Cerebellar Lobe (twenty-five cases):

1. Susceptibility to ear stimulation absent in all cases.
2. Past pointing after stimulation very poor in practically all cases with the exception of five, where there was only a fair past pointing.
3. Vertigo definitely diminished in all of the cases, in some almost to the vanishing point.
4. Pelvic girdle movements poor in all cases; in the vast majority very poor.
5. Spontaneous vertical nystagmus definitely present only in thirteen cases: suspicion in six, and absent in six.
6. Nystagmus after turning increased in about one-half of the cases; diminished in the other half.
7. Vertical canals' function affected in half the cases; normal in the others.
8. Horizontal canals normal in half the cases; perverted in the others.

Midline Cerebellar (six cases):

1. Susceptibility to ear stimulation absent.
2. Past pointing prolonged and fair in two: poor or absent in four.
3. Spontaneous vertical nystagmus present in all.
4. Pelvic girdle movements poor in all.
5. Nystagmus after turning exaggerated.

6. Vertigo normal in two, reduced in the others.
7. Vertical semicircular canals affected in all.
8. Horizontal canals' nystagmus perverted in three.

In cases where the pons is involved, there is a marked spontaneous nystagmus in all directions. The horizontal canals' responses are apt to be markedly impaired, not perverted, and dissociation of eye movements following stimulation is present.

Cerebellopontile Angle Tumors (thirty-seven cases):

1. Susceptibility to ear stimulation absent.
2. Spontaneous past pointing absent.
3. Vertical spontaneous nystagmus present in practically all of the cases.
4. Pelvic girdle movements poor in all cases.
5. Nystagmus after turning markedly diminished, but unequally as compared with turning to the right and turning to the left.
6. Vertigo markedly diminished, but unequally so as compared with turning to the right and turning to the left.
7. Vertical canals' responses all absent, with the exception of two cases, where the responses were normal in one, and very slow in the other.
8. Horizontal canal usually shows perverted nystagmus on side opposite the lesion.
9. Total loss of function, cochlear and vestibular, on the side of the lesion.

Cerebellopontile Angle Tumors Secondary to Cerebellar Involvement (four cases):

The notable difference in the responses obtained in this type of lesion, as compared to the cerebellopontile angle lesions is that the vertical canals on the side opposite the lesion show some response in every instance. In addition, all of these cases have evidence of cerebellar involvement, in that the vertigo is very short and the past pointing after stimulation unusually poor.

Differential Diagnostic Signs Between the Various Areas.

A careful analysis of these various "phenomenon-complexes" will show that there is enough individuality in these group findings for the localization of lesions—marked in some, and less marked in others, but present frequently enough to enable the experienced examiner to arrive at a diagnosis. In the examination of patients with the vestibular tests, one encounters the same

difficulties as would be encountered in an examination of these patients by any other method. Neurologic syndromes are also apt to vary and overlap. The first step in the diagnosis is to determine whether the lesion is supra- or subtentorial. The vestibular group findings for such a differentiation is quite definite. Having then placed the lesion, let us say supratentorially, one can take the frontal lobe complex as a standard from which to differentiate lesions located elsewhere. An occipital lobe tumor is identified by the definitely prolonged and exaggerated past pointing out of proportion to the vertigo after stimulation, and the absence of susceptibility to the tests. In lesions at the junction of the parieto-temporo-occipital lobe there is an impairment of all the vestibular fibers from the ear on the side opposite the lesion, and the patient is not susceptible to stimulation. In lesions of the motor area, the pelvic girdle movements are poor, the vertigo after turning is apt to be unusually prolonged, especially if there is any extension of the lesion toward the temporal lobe, and there is very slight if any susceptibility to stimulation. In a suprasellar lesion, the pelvic girdle movements are poor, vertical semicircular canals, if interfered with at all, are usually affected on one side only, and vertigo and nystagmus are out of proportion to each other. In pituitary lesions the vertigo after turning is usually shorter than the nystagmus, the vertical semicircular canals' responses are usually not interfered with, and susceptibility to stimulation is very apt to be absent, or if present, it is exceedingly marked.

In differentiating the various subtentorial lesions, the cerebellopontile angle tumors present the most definite complex, namely, the loss of all responses on one side, including hearing, with a loss of the vertical semicircular canal's function on the opposite side, whereas in the cerebellar lobe lesions, both eighth nerves show presence of function with the other characteristics, namely, markedly impaired vertigo and past pointing, poor pelvic girdle movements, and loss of susceptibility to stimulation.

Wherever a difficulty arises between the clinical diagnosis of a suprasellar and cerebellar lesion, they can be differentiated by the vestibular tests as follows: a suprasellar lesion shows fair vertigo and past pointing after stimulation and no spontaneous vertical nystagmus. Furthermore, these patients always are somewhat susceptible to ear stimulation, whereas a cerebellar lesion would exhibit just the opposite findings, such as definitely

impaired vertigo and past pointing, well developed spontaneous nystagmus and total immunity to ear stimulation.

Conclusions.

1. A brain lesion usually shows abnormal vestibular test findings.
2. Generalized increased intracranial pressure, as such, cannot be diagnosed by the vestibular tests.
3. The vestibular findings indicative of the laterality of a lesion are definite and reliable, when present, but not all brain lesions exhibit these signs.
4. The vestibular tests can usually differentiate definitely between a supra- and subtentorial lesion.
5. Each anatomic area presents vestibular group findings of its own.
6. While in the vast majority of cases the diagnosis of brain tumors can be made by the vestibular tests, their value would be the greatest when taken in conjunction with the general clinical findings.

DISCUSSION.

Dr. Lewis Fisher, Philadelphia, Pa., (closing): I am not going to present any extensive rebuttal, except to call attention to the fact that the subject matter of the paper presented is not the result of deliberate theorizing. Dr. Glaser and I did not sit down together and figure that given a lesion here or there, the response must be such and such. The matter presented in the paper represents the findings in actual cases as proven at autopsy and operation. Unfortunately, the number perhaps is not so large as it might be. These results were just as much a surprise to us as they are perhaps to Dr. Sachs. When we were thru with the analysis and tabulation of the vestibular responses, it was a revelation to us to find that there was this definite grouping of the responses in the various lesions presented.

RECENT DEVELOPMENTS IN NEUROLOGY AS IT APPLIES TO THE EYE, EAR, NOSE AND THROAT: THE NOSE AND THROAT.

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The results of academic research of medical topics can be of practical value only by correlation with pathologic and clinical manifestations, and the ever changing attitude towards former theories and practices results not only in progress but makes our studies fascinating and picturesque.

A survey of recent developments in any branch of medicine should deal with original reports of important discoveries, discussions of old and moot questions, proved improvements over accepted views, and a solution of hitherto unsolved problems.

The function of the reporter of this survey, I take it, is properly to evaluate these reports and discussions and present them to you in concise and concrete form, with but little personal comment. The work of others thus becomes a stimulus and a guide to our own efforts.

Bearing all this in mind, I have omitted much that has appeared in the literature and have utilized the reports of unusual cases only when some important fact might be noted.

THE NOSE.

CEREBROSPINAL RHINORRHEA: Our knowledge of this ever interesting subject is increased by Locke's[1] study and report of cases, in which he states that obstruction to the circulation of the cerebrospinal fluid and increased intracranial pressure are the two necessary factors in the formation of a fistula between the anterior cranial fossa and the nose, the commonest lesions being cerebral neoplasm and inflammatory or congenital obstruction.

Caliceti[2] reports a new oculoendocranial syndrome of nasal origin manifested by headaches and diplopia. Nasal obstruction and paralysis of the abducens were found on examination. Intranasal operation resulted in cure. In explanation, the author stresses the relation of the veins entering the cavernous sinuses

from the pterygoid plexus, and the relation of the abducens to the cavernous sinus. Polypoid hypertrophy of the middle turbinate causes venous congestion, resulting in compression of the nerve as it passes thru the cavernous sinus.

Stimulating the study of disorders of olfaction, the classification by J. H. Kenneth[3] is of much value. PATHOSMIA may be divided into:

1. Cryptosmia, odors not preceived, but olfactory apparatus intact.
2. Anosmia, complete loss of smell.
3. Hyposmia, diminished olfactory acuity.
4. Hyperosmia, hyperesthesia of smell.
5. Merosmia, perception of certain odors but not of others.
6. Heterosmia, perception of other smells than those presented.
7. Autosmia, subjective sensations of smell; hallucinations.

FOCAL EPILEPSY in chronic sphenoidal sinusitis is reported by George A. Hyslop[4]. Operation on the ethmoid and sphenoid showed necrosis. The local symptoms were relieved and there was no return of focal disturbance of the brain.

LEPTOMENINGITIS following operation on the nose and ethmoid is reported by A. Logan Turner and F. Esmond Reynolds.[5] The article is of especial importance in showing the pathway of infection. It shows that following operation on the nasal cavities and ethmoidal cells, purulent infection of the perineural sheaths of one of the olfactory nerve filaments took place. By direct upward extension, this gave rise to primary acute purulent leptomeningitis.

CHOKED DISC AND RETROBULBAR NEURITIS: Whether we accept the statement of Cushing[6], that choked disc is not produced by postethmoidal or sphenoidal disease but is present only on increase of intracranial pressure, or that of Sluder[7], that pronounced swelling of the nerve may follow diseases of the sinuses, the fact remains that not a few cases are benefited by sinus surgery as shown by Meller and Hirsch[8] and others. Retrobulbar neuritis may be due according to these authors to sinus disease when few if any symptoms are seen in the nose. They insist that microscopic examination of tissue removed is necessary and may show only slight changes. The inflammation can pass by way of the bone marrow spaces to the optic nerve. Characteristic of the disturbance are the acute commencement, the quick progress and the unilateral lesion.

Ruskin[9] classifies NEUROGENIC DISTURBANCES arising from infection of the sinuses into:

1. Acute pains and inflammation of the sinuses.
2. Chronic neuralgia.
3. Vasomotor and secretory states.
4. Systemic effects.

TIC DOULOUREUX is due to involvement of the V and VII nerves thru the nasal ganglion, which accounts for the good results that may be obtained from treatment of this ganglion.

White[10] makes a plea for conservatism in the treatment of OPTIC NEURITIS. Routine opening of accessory sinuses is unnecessary, but surgical intervention is recommended when loss of vision is total. Eighty-five per cent get well under conservative treatment. Patterson[11], of Edinburgh, further elucidates this question by stating that retrobulbar neuritis of the chronic persistent type is cured or relieved by free operation, claiming that the acute cases generally get well spontaneously, contraindicating the need of operation in the early stages.

Interest continues in the study of SLUDER'S SYNDROME, namely pain from the ala nasi to zygoma, to back of the ear, and down the neck, shoulders and arms.

Weisenberg[12] believes that in many instances the results of sphenopalatine injection are unsatisfactory, but that injection of, or radical operation on the V nerve is necessary for elimination of the pain. In other instances, Weisenberg believes that inflammation of the IX nerve without relation to the nasal ganglion is responsible for the pain. He states that the IX and V nerves supply contiguous sensory areas, and that it is conceivable, that in an inflammation of either, pain may be present in the parts supplied by both, i. e., in the lower part of the face, tongue and throat. It is well to remember, as Weisenberg points out, that instead of pain, only vasomotor and secretory symptoms may be found. Socalled hay fever manifestations may be relieved by injection of the sphenopalatine ganglion.

THE LARYNX.

ANATOMY: Bellucci[13] states that the clinical problem of LARYNGEAL PARALYSIS of purely cortical origin is not definitely solved as yet, and demonstrates by animal experiment the complete disassociation of the phonetic and respiratory functions of the larynx. A. Brown Kelly[14] emphasizes that the chief respiratory centers are in the medulla, the subsidiary ones being in the

cortex, while the principal phonation centers are cortical and the less important, bulbar. A cortical lesion, therefore, is not capable of producing complete paralysis of a vocal cord.

The anatomic studies of Dilworth[15] are still outstanding as authoritative on the distribution and anastomoses of the laryngeal nerves, and it is especially noteworthy, as bearing upon our clinical observations, that there is a very close anastomosis between the superior laryngeal and inferior laryngeal nerves, and that the superior sends a branch to the muscle in the aryepiglottic fold and the arytenoideus.

Bender[16] has shown that the cerebellum has some control over the motor activities of the vocal band, but this activity is not localized to any particular spot.

Rawson[17] in writing on GERHARDT'S SIGN, a laryngeal syndrome characterized by an intense dyspnea with integrity of the voice, contends that there are separate nuclei which preside over the function of each muscle or muscular group. Negus[18] explains the vulnerability of the abductor group on the theory that adduction is an earlier function than abduction, and therefore the power of the former survives the power of the latter.

ETIOLOGY:

PARALYSIS OF THE LEFT RECURRENT DUE TO MITRAL STENOSIS: Reiche[19] shows that this is produced by dilatation of the left auricle compressing the pulmonary artery, with adhesions to the aorta and the left recurrent nerve, causing grey degeneration and paralysis of the nerve. Notkin[20] shows the most frequent cause is thru a dilatation of the pulmonary artery, or by pressure transmitted by the left auricle and the pulmonary vein.

Paralysis of the left recurrent nerve was shown by Julich[21] to be due to SCLEROSIS OF THE AROTA, without aneurysm or mediastinal tumor.

Attention is called by Lugli[22] to the large percentage of laryngeal lesions in TABES, at or before the ataxic stage, sudden aphonia or dyspnea indicating involvement of the X and XI cerebral nerves and their nuclei.

No more comprehensive article on the effect of TRAUMA on the laryngeal nerves has appeared than that of Judd, New and Mann[23] published in 1918. This is of importance, because of the frequent involvement of the nerve following thyroidectomy. Section and ligation of the nerve causes complete and permanent paralysis. Stretching does not impair the function of the vocal band. Stretching for a long time impairs the function, due more

to operative trauma than stretching itself. Pinching with the hemostat impairs the functions temporarily.

GOITRE as a cause of recurrent paralysis according to Zeno[24] occurs in from 7 to 35 per cent. It is nearly always unilateral, and when bilateral probably malignant. Mullin[25] believes that laryngeal paralysis is the result of goitre only when the latter is malignant. "A Swiss Colleague," quoted by St. Clair Thomson[26], bears this out. (Professor Nager[27]) St. Clair Thomson himself states that in England there are cases on record of paralysis existing beforehand and disappearing after a successful operation on the goitre. The intrathoracic goitre, according to A. Brown Kelly[28], is more apt than the cervical variety to cause pressure on the nerve. Temporary immobility might be observed, according to Nager[29], by sudden enlargement of the goitre, as for example from hemorrhage. Birkett[30] is convinced that paralysis of the vocal cord is not necessarily a sign of malignancy.

An interesting contribution to the etiology of HOARSENESS in children depending upon vocal cord paralysis was published by Taro Matsui[31] as occurring in Japan where beri-beri is prevalent. This condition occurs in children nursed by mothers or nurses suffering from beri-beri, the condition being cured when the nursing is discontinued. The left recurrent laryngeal is more frequently involved, the explanation being that it is a longer nerve than the right and the analogy being that the disease attacks the nerves at the sole of the feet first because of their length.

PARESIS of the upper eye lid with CONTRACTED PUPIL also spoken of as the CLAUDE BERNARD-HORNER syndrome is a new and interesting observation by Villard[32] following injection of alcohol into the superior laryngeal nerve.

DIAGNOSIS: Of diagnostic value is an unusual combination of NYSTAGMUS WITH RECURRENT LARYNGEAL PARALYSIS, mentioned by Cohn[33] in a case of syringobulbia and in another of multiple sclerosis, and Stein[34] believes that the diagnosis of a lesion in the medulla can be made when this combination is present.

A diagnostic caution as voiced by Roger and Reboul-Lachaux[35] is offered in a report of a case of a DEFORMED PALATE with RECURRENT PARALYSIS the latter only due to aortic aneurysm suggesting a central lesion involving the glossopharyngeal and hypoglossal.

A method for the clinical demonstration of recurrent paralysis by Stockert[36] is performed by placing a stethoscope between the manubrium and the body of the sternum during the phonation of

vowels, when a coup de glotte can be distinctly heard in the normal. In the case of recurrent paralysis, the vowel is preceded by a distinct "H." This may be of interest but is of no practical value.

PROGNOSIS: According to Proby[37] laryngeal paralysis is curable when due to:

1. Poisoning by lead, arsenic, sulphur, alcohol, atropin, morphin, cocain.

2. Infectious disease such as diphtheria, typhoid, typhus, influenza, cholera, malaria, profuse blennorrhea, puerperal fever, jaundice.

3. Tuberculosis, frigorism, syphilis.

TREATMENT: Operative treatment following the suggestions of Jackson (*cordectomy*) and Frazier (*nerve anastomosis*) seems to be attracting more or less attention. Several new methods are here mentioned not because they have been definitely proved of value and universally accepted but more especially because of their uniqueness and possibilities.

Rawson[38] states that the operation of Moore offers excellent recourse in the paralysis of abduction. By it the cord is displaced and shortened anteroposteriorly, raising it one-quarter of an inch. The operation fixes the cord in complete abduction thus overcoming the respiratory difficulty.

For the restoration of the voice in unilateral paralysis, Kretschmann[39] injects paraffin into the paralysed cord, which transforms it into an abutment which the unparalysed cord approximates in phonation.

Nikolajew[40] shows that it is possible, by a difficult technic however, in experiments upon dogs to implant nerve ends, after section, into muscles, thus successfully inervating them.

Max Grassmann[41] suggests one of two operations; that of Payr or that of Schmerz. Payr excises a flap of thyroid and pushes it towards the interior of the larynx. Schmerz shortens the transverse diameter of the larynx by compressing the wings of the thyroid cartilage.

None of these operations seem to have been generally adopted except by the inventors and one or two adherents, nor has there been any marked improvement or changes in the neurologic aspect of treatment except as purely secondary to the advance made in the management of central nerve lesions.

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CORRELATION OF PAPERS IN SYMPOSIUM ON NEUROLOGY OF EYE, EAR, NOSE AND THROAT

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Your president asked me a year ago to take part in this symposium and told me my function would be correlation. According to Webster's dictionary, correlation means showing the relationship between things. I have gone over the three papers you have just heard with this in view, but find that there is little to correlate, as each essayist has treated his subject from a different angle; and then it occurred to me that your President selected a neurologic surgeon for this because he wished to have the relationship between neurologic surgery and neuroanatomy, laryngology, and neurootology brought out. I trust my interpretation of my function is correct.

Some years ago, I had the pleasure and privilege of addressing you at your Philadelphia meeting, and on that occasion made a plea for a more intimate association between these various specialties. The fact that you have this symposium today indicates perhaps that this suggestion was looked upon with favor—that we specialists must get closer together.

Dr. McDonald has brought out in his paper the great value to the neurologic surgeon of a careful study of the eye fields. Nothing in our diagnostic armamentarium has been of greater value than a careful study of the eye fields. As Dr. McDonald has pointed out, whereas formerly the temporal lobe, especially the right lobe, was a silent area, since we have carefully studied the eye fields it has been taken out of that category. One point of great interest and importance that Dr. McDonald did not speak of, but I trust he will refer to in his closing remarks, is whether he believes the macula has bilateral representation. My own clinical studies have led me to believe that it has.

In drawing attention to and dwelling so fully on the recent work of Brouwer, Dr. McDonald has made one thing quite clear, it seems to me; that the ophthalmologist must find time to make much more careful studies on eye fields than he has in the past.

The usual way of taking eye fields is totally inadequate. Rarely has an eye field been sent to us that we have not had to repeat, because the ophthalmologist as a rule takes the field every thirty to forty-five degrees, whereas it should be taken every fifteen degrees.

In view of my interpretation of my function, I shall take up only those phases of Dr. Levy's paper that bear on the field of neurologic surgery. The very first subject he mentions is of enormous interest—spinal rhinorrhea. This occurs most commonly in cases of pituitary tumor and in traumatic conditions. Its greatest importance is the ever present danger of the patient developing a meningitis. Usually the rhinologist sees these cases first, and if he does not recognize that he is dealing with a cerebrospinal fistula, very slight manipulation in the nose may cause a disaster. A meningitis that is introduced thru the nose directly into the subarchnoid space is, in my experience, always fatal. No operative procedure thus far has been of any value in stopping such a meningitis. With a very thin cribiform plate, operations on inflammatory processes in the nose are of equally great danger. As I have seen some of the end results, it seems quite in order to sound a note of warning. In the past two years, I have seen six cases of meningitis with a fatal outcome which followed directly on a nasal operation. In practically all of these cases, there was an acute inflammatory process in the nose, and I have wondered whether the same principle that the general surgeon applies might not very properly be considered in these cases, namely, that an acute inflammation ought not be operated on unless there is definite walling off, or abscess formation. Is not the acute condition in the nose comparable to that of a cellulitis, which no good general surgeon would ever think of incising?

The attempt by Kenneth to differentiate various disturbances of olfaction may lead to important advances in the diagnosis of involvement of the first cranial nerve. It has seemed to me, however, that the elaborate terminology he suggests gets us no nearer our goal. Such work as Proetz has been doing here in St. Louis seems of far greater interest and value; for up to the present time, we have no means of differentiating central from peripheral loss of smell, and his work may make this possible.

The question of focal epilepsy has always been a problem with which the neurologic surgeon has occupied himself a great deal. Whether Hyslop is correct in claiming that chronic sphenoidal sinusitis is an etiologic factor is distinctly open to ques-

tion. Anyone who has occupied himself much with epilepsy would hesitate, I think, to accept this view, since focal epilepsy beyond a question, I believe, is always due to an irritation of the cerebral cortex, but many epileptics temporarily stop their attacks after all sorts of operative interference—which does not prove, however, that the operative interference actually dealt with the condition.

A careful study of Hyslop's case report makes me feel that he has not excluded either a brain tumor in the temporal lobe or an abscess in that region. Furthermore, I would want to know how carefully the eye fields had been examined. A temporal lobe lesion produces an eye field defect which may readily be missed unless the examiner examines every fifteen degrees. Sometimes the defect in a temporal lobe lesion occupies only fifteen degrees.

The occurrence of eyeground changes, choked disc, with inflammatory processes in the nose, is a question about which every neurologic surgeon has very definite views, and tho I am not willing to be quite as drastic as Cushing and say that it never occurs except with increased intracranial pressure, I am convinced that in the vast majority of cases it occurs only with increased intracranial pressure. I have seen a great many cases of brain tumor in which valuable time was lost because repeated nasal operations had been done, with the idea that the choked disc was due to some infection in the nose, and I think it is sound medicine to insist that every case of choked disc should be considered as due to an intracranial lesion until proved otherwise. It was before this very Society at its Philadelphia meeting some years ago, that Professor Van der Hoeve, of Amsterdam, made the statement that he was convinced in the majority of instances it is impossible to tell from the appearance of an eyeground whether one is dealing with a choked disc or an optic neuritis, and he believed, as he told me in a personal communication at that time, that choked disc was always due to increased intracranial pressure.

The classification of neurogenic disturbances by Ruskin, to which Dr. Levy refers, brings up another subject of great importance in which the neurologic surgeon is deeply interested, namely, tic douloureux. True tic douloureux is always a disease of the V nerve, and I have never seen a case in which it was due to disease of the VII nerve as Ruskin states. This seems to be absolutely impossible, since the VII nerve is a motor nerve.

The difficulty we frequently meet with is to make the correct diagnosis, as the numerous cases of pain in the face that the rhinologist so frequently encounters offer many obscure problems. Unless the pain is confined to the distribution of the V nerve and does not spread to the other side, one is not justified, I believe, in making a diagnosis of tic douloureux. By adhering to this attitude and occasionally testing out a case with an alcohol injection of the second or third division, unnecessary operations on the Gasserian ganglion may be avoided. Weisenburg's point, that disease of the glossopharyngeal nerve simulates tic douloureux at times, is unquestionably correct, but if one keeps in mind that in glossopharyngeal neuralgia the pain is usually referred to the pharynx, errors will be avoided.

Whether A. Brown Kelly is correct in assuming that there are respiratory centers in the cortex is open to question. The experimental work by Victor Horsley, Sir Felix Semon, Sherrington, and others does not indicate that there are such centers anywhere but in the medulla. Sir Felix Semon did prove beyond all doubt that there is a center for phonation near the foot of the central fissure, but he never showed that there was any control of respiration.

There are numerous other subjects that Dr. Levy has referred to which, if time permitted, I should like to say something about, but the paper by Dr. Fisher and Dr. Glaser is of such great importance to the neurologic surgeon, that I must take it up now.

As we all know, nobody in this country has done more for neurootology than Dr. Fisher. Perhaps it is not too much to say he is neurootology, and his enthusiasm and earnestness have done much to advance this field. However, I think Dr. Fisher and Dr. Glaser go too far when they attempt to diagnose lesions in the various portions of the cerebral hemisphere and the pituitary region by the vestibular tests, and when they say in their conclusions that in the vast majority of cases the diagnosis of brain tumors can be made by the vestibular tests, I must take issue with them. I have gone over very carefully the symptoms that they ascribe to the various portions of the cerebrum. There are eight different symptoms under each one of these headings that they have discussed.

Frontal lobe lesions differ from all others in that there is marked susceptibility to ear stimulation and, whereas this is very interesting, no neurologist, and certainly no neurologic surgeon, would venture to operate on a patient's frontal lobe on such

meager evidence. The outstanding characteristic of the motor area is said to be poor pelvic girdle movements, and that vertigo is apt to be unusually prolonged.* Now, to my way of thinking, if vertigo is unusually prolonged, it is an indication that the patient is susceptible to the tests, therefore right here there is, it seems to me, an inconsistency in the author's reasoning. Dr. Fisher has not made clear what he means by susceptibility to the tests. If he refers only to nausea and vomiting, he ought to make that point clear, but it seems to me that if a patient has increased vertigo, it is just as much an evidence of susceptibility as nausea and vomiting. Furthermore, pelvic girdle movements being poor are certainly a much less certain diagnostic sign than a partial or complete paralysis of part or all of the opposite part of the body, which is an almost universal accompaniment of a tumor in the motor area. In suprasellar lesions, the pelvic girdle movements are said to be poor and vertigo and past pointing are out of proportion to each other, but I would like to point out that suprasellar lesions not only may involve the frontal lobe, the temporal lobe, but also the basal ganglia thru which the fibers of the pyramidal tract run. In the first place, the anatomic pathways of the vestibular mechanism have never been completely worked out. In the second place, both Barany and the American neurootologists have completely ignored the fact that is well known to all neurosurgeons, that in practically all posterior fossa lesions there is a dilatation of the lateral and third ventricles. Such a dilatation must inevitably exert pressure on the pathways in the pons. In 1920, Dr. B. Y. Alvis and I presented a paper before the American Neurological Association on the anatomy and physiology of the vestibular portion of the VIII nerve, and on that occasion we emphasized this point. I am absolutely convinced that many of the disturbances of the vestibular mechanism that the neurootologist finds are due to this associated internal hydrocephalus, and are, therefore, more the signs of general intracranial pressure than of localizing value. It is rather interesting in this connection to note in the report of Dr. Fisher's and Dr. Glaser's paper, that the farther back the lesion occurred in the

*In Dr. Fisher's paper that he submitted to me, under the sixth heading under "motor area," on page six of his manuscript, the statement is made "vertigo after stimulation definitely diminished in all," while on page eleven of his manuscript, under the heading "differential diagnostic signs between the various areas" the statement is made, "in lesions of the motor area. . . . the vertigo after turning is apt to be unusually prolonged and there is very slight if any susceptibility to stimulation."

cerebral hemisphere, especially in the occipital lobe cases, the more were the vestibular functions interfered with, and it is a well known fact that the farther back in the cerebral hemisphere a lesion occurs, the more frequently does an internal hydrocephalus occur. I have never seen an occipital lobe lesion in which this was not the case. For this reason, in the last eight or nine years we have made very much less use in my clinic of the vestibular tests than in the early days. The vestibular tests are certainly invaluable in recognizing a tumor of the VIII nerve.

Another point that has seemed of great importance to us here, is that the great susceptibility which Dr. Fisher and Dr. Glaser emphasize of patients with supratentorial lesions is a distinct danger. To bring on vomiting in a supratentorial lesion can seriously jeopardize subsequent operative procedure on a patient, and whereas we all are greatly interested in the advancement of science, I do not believe that any of us are willing to subscribe to the principle that was uppermost in the mind of Barany when he was in this country, that science comes first and the patient second. The neurologic surgeon welcomes any addition to his diagnostic armamentarium, and wherever these tests hold out a prospect of any help, they should undoubtedly be used, but two points, it seems to me, must always be kept in mind: That the vestibular nerve is only a part of one of the twelve cranial nerves, and secondly, that those pathways of the vestibular mechanism that we know, lie in the floor of the fourth ventricle, where they are readily affected by any changes in pressure from an internal hydrocephalus.

TRANSACTIONS

OF THE

THIRTY-THIRD ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OPHTHALMOLOGIC SESSIONS

THE DIAGNOSIS AND CLASSIFICATION OF CATARACT IN THE ADULT.*

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The diagnosis and classification of cataract in the adult has been simplified within the past few years by the clinical introduction of two instruments, the slitlamp and the camera. The former has so illuminated the paths of the investigator that the reflected light has made all clinicians brighter. Photography of the lens is on a firm footing, and our daily observations prove the necessity of this form of record.

Symptoms suggesting a possible diagnosis of cataract, such as dimness of vision, influenced by light and shadow, second sight and polyopia, are too well known to need comment.

The physical signs must depend upon the type, the extent, the actual stage of development, and above all the anatomic position of the opacity.

The use of a mydriatic in all eyes in which no contraindication exists, is not only desirable but absolutely essential. The drug most commonly used is homatropin, grain 1 to ʒ 1. This is instilled after the primary ophthalmoscopic examination and following tension determination by fingers. Sometimes we use cocain, less often euphthalmin, dubosin or atropin. The size of the pupil must be recorded as part of the findings. After the drug has acted, our routine consists of oblique illumination with or without a loup, depending upon the conditions found. This method is of great importance when the lens clouding is advanced or so localized as to afford contrasting shades. The ophthalmoscope with a +20.00 is then used, by carefully focusing all visible portions of the lens. After this, we view the eye with a plane mirror at a meter distance, and finally check with the slitlamp and take a picture. Too little attention has been paid

*Stereoscopic photographs were used to demonstrate the numerous forms of cataract found in the adult.

to the meter distance study. It is the most valuable method of outlining the opaque portions as dark areas against the pink background, sometimes quite difficult with all other tests. By this established habit, there is no probability of overlooking even minute microscopic defects.

The slitlamp, when used with a wide band at an obtuse angle, brings into relief the adult nucleus surface, the involved regions anterior and posterior to this layer, as well as most other parts of the lens. When the examiner is once orientated, a narrow, brilliant beam accurately focused on the affected part makes microscopic study possible. The obtuse angle must be maintained for the anterior opacities, but an acute angle is better for posterior lens inspection.

All are familiar with the usual complex structure of the lens, including the differentiated, embryologic, fetal, infantile and adult nucleus, the cortex and the subcapsular lines.

For our discussion, we consider cataract in the adult to mean one that has developed after the adult nucleus has been laid down. My fellow reporters are, I am informed, to consider the other varieties of cataract, and even at the risk of leaving some unusual forms out of the symposium, I will resist temptation and present only this one group.

A cataract in an adult may remain stationary, progress very slowly, or rapidly pass thru the various stages to maturity. It is because of the first two groups, that nostrums have been given wide publicity and reputable ophthalmologists have looked with angry discontent on those who exploit the sick. To differentiate these forms is possible only by the complete, careful analysis following the examination details previously described. Any opacities of the lens fibers distal to the adult nucleus are, therefore, cataracts in the adult, altho, strictly speaking, capsular opacities may by reduplication or imprint involve these layers.

At the present time, amid the changing views of vascillating opinions, we consider coronar and dilacerate forms as forerunners of complete opacification and believe nuclear, posterior saucer, cuneiform and dust like types definitely adult. By means of the stereophotographs, each division is visualized, for the terminology rests on the location and the physical characteristics of the lens. With these forms well understood, we may scrutinize the slitlamp details of the typical progressive cataract.

The earliest sign is a fluid filled cleft appearing as a dark optically empty line or narrow thin wedge. The fluid becomes

cloudy, the individual globules distinguished by their light reflecting surfaces, and soon the enclosing walls become gray, at first microscopically, but without much delay the increased density makes the opacity visible. We note the number, depth, width and position of these clefts, which follow no anatomic lines or divisions, so that they exhibit the bizarre forms so frequently seen in practice. They are, however, most numerous in the anterior cortical layers, or in the anterior adult nuclear fibers in the inferior nasal sector. It is strange that these isolated triangular opacities are not only the commonest, but also among the slowest to develop, often remaining almost the same for decades.

If the lens sutures are separated, vacuoles are seen. A single globule or, later, a complete subcapsular fluid layer may be evident. The advancing changes consist of the breaking down of lens fiber, with liquefaction and opacification. One type overlaps another, or, as often happens, one form may be completely overshadowd by another. This is illustrated in the peripheral adult nuclear type, in which a posterior cortical development is started, or in the nuclear one, where the entire lens eventually becomes opaque. The invasion of new zones, the liquefaction of some areas and the grayness of others makes each cataract an entity altho susceptible of grouping. The progress is exemplified in the hypermature stage, where liquefaction continues until the nucleus lies in a cloudy fluid containing bright crystalline flakes, and the capsule becomes thick, dirty gray—a Morgagnian cataract. Another hypermature type is that in which the fluid is absorbed and the lens becomes thinner and denser. This form may be anticipated when rounded white spots are seen just beneath the capsule.

The coronar progressions are seen in the series of stereos, where the crown of peripheral finger like extensions are even tually obscured by the growth of each single finger and the involvement of adjacent lens fibers until finally opacification is complete. It is impossible to be certain of the exact form which preceded the general opacity, altho the visual history may give a valuable clue, that is poor near sight in the earlier stages of cortical cataract and indistinct far sight in the nuclear changes. The advancement of the dilacerate type is also demonstrable. Stellate, floriform and coraliform groups are also presented.

The adult is predisposed to complicate cataract by reason of his liability to uveal tract inflammations and constitutional inva-

sions. This form starts with a massing of spherical, almost bubble like, elevated, clouded spheres in the posterior cortical layers. These extend into the cortex and are distinguished by their thickness and iridescence from the ordinary adult posterior saucer cataract, which looks like a sheet in which holes, darker spots, have been punched. A complicate cataract is frequently accompanied by gross evidence of the comitant features of the ocular disease.

The secondary cataracts, such as anterior capsular opacities, need only to be mentioned to be visualized and understood. The after cataract picture of glistening glass globes or frog spawn described by Elschnig is a distinctive type of this postoperative condition.

Summary.

Certain types of opacity appearing in the lens of patients beyond middle life may be likened to the graying of hair: they seem to be associated with maturity, and altho demonstrable and too often vision reducing, they practically never cause blindness, are uninfluenced by treatment and should not be described as cataracts to the apprehensive patient.

Various changes in the clearness of the lens seem to be forerunners of general opacity, particularly the coronar and dilacerate groups.

A large class of progressive cataracts are the cuneiform, the dust like, the posterior saucer layer and the nuclear opacities.

As yet no treatment has been discovered to restore clearness or bring back to usefulness the opaque lens fiber. Any claim of this has not been substantiated by photographic proof. Visual function is not a guide as to the process transpiring within the capsule. The act of vision is a complicated mechanism and the clarity of the lens is only one of the many factors involved.

DISCUSSION.

Dr. Conrad Berens, New York City, N. Y.: Such an instructive paper concerning the timely subject of the microscopic diagnosis, classification and recording of pathologic changes in the senile lens brings up several points, which are so important, that they may possibly bear reemphasizing, even tho one could find nothing new to add to such a careful and complete study.

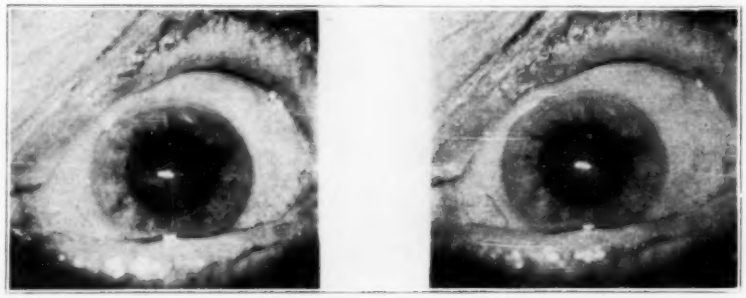
The slit lamp is undoubtedly a valuable aid in diagnosis, but we should not forget that it has taught us to note some changes by simpler methods which are particularly of value in the study of the adult lens. A very useful slit beam can be made with the handle of the May, Bausch and

Lomb and De Zeng ophthalmoscopes, while the Cameron ophthalmoscope is furnished with a special head for slit and dot beam illumination. The Keeler model also gives a fine dot beam, all these methods of illumination thus making study of the lens with a binocular or ordinary loupe a valuable clinical procedure. It should be unnecessary to say that complete dilatation of the pupil is essential for accurate diagnosis with the slit lamp, the ophthalmoscope, and the anterior segment camera.

It has always seemed that the lens opacities could be seen best with a plane mirror at 1 meter, and then studied with a +4.00, 5.00 or 6.00 D lens in the ophthalmoscope used at the focal distance, with a well concentrated altho not too brilliant beam of light.

Photography, and particularly stereoscopic photography, has given us a valuable method of recording findings, and even with Dr. Hardy's and my technic, which is not as perfect as Dr. Bedell's, has proved most satisfactory for purposes of record and for teaching. It is hard to understand why such a valuable method of recording the effects of treatment of pathologic changes in the lens has not been generally used.

We agree that no treatment yet devised will restore complete transparency to a lens fiber which has undergone advanced pathologic changes. With the use of dionin and lens antigen alone[1], lens changes progressed and vision diminished in eight of nine cases studied. It has not seemed justifiable to treat others by either of these methods to the exclusion of general medical treatment, which in some cases seems to keep the lens



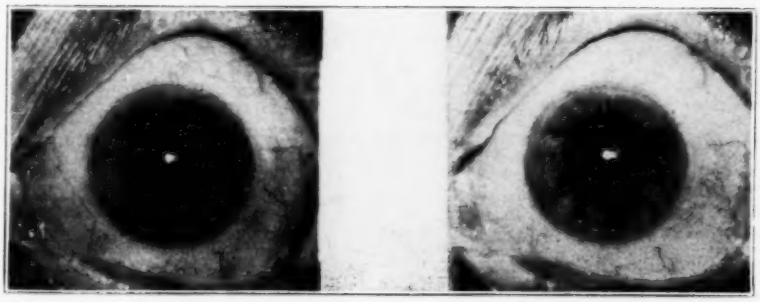


Fig. 1. Miss J. A. T. Left lens 5/22/28. Compare with Figure 2.
Fig. 2. Miss J. A. T. Left lens 10/8/28. Compare with Figure 1. Little, if any, change in lens in spite of fluctuating visual acuity.

changes from rapidly increasing, and certainly improves the vision in some patients, at least temporarily. The improved vision may be attributable in part to increased transparency of the lens, but improvement in the retina and centers for vision has also been a big factor in some of the cases observed.

To illustrate the fact that vision can improve and fail altho lens pathology is apparently unchanged, two stereoscopic photographs of Case 1 are shown. (Figs. 1 and 2.)

(Case 1.) Miss J. A. T., aged 70; first seen 4/1/25. Best corrected vision 20/40 R. E. and L. E.

Diagnosis: Incipient cataract, O. U. involving the pupillary area. 6/16/25, V. R. E. 20/30 + 2; L. E. 20/50 + 2.

1/6/26, V. R. E. 20/30; L. E. 20/20, after general hygienic treatment, the extraction of two teeth with apical abscesses and the local use of massage and dionin. No apparent change in lens pathology.

1/28/27. V. R. E. 20/40; L. E. 20/60 + 2. No increase in lens changes.

5/18/28. V. R. E. 20/30; L. E. 20/30. No increase in lens changes.

10/4/28. V. R. E. 20/50; L. E. 20/50. Ophthalmoscopic examination: Apparent slight increase in lens changes.

10/10/28. Vision improved with careful refraction to R. E. 20/30 — 2; L. E. 20/40 + 1.

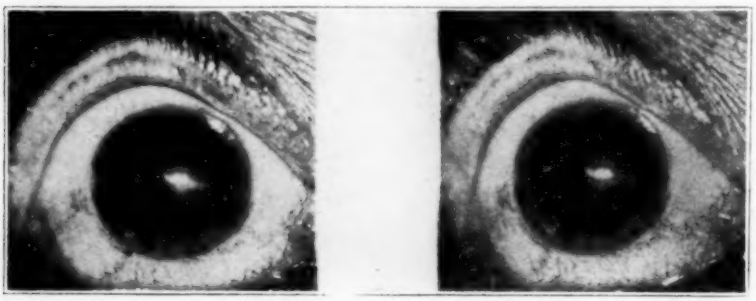


Fig. 3. Case 10467. N. Y. Eye and Ear Infirmary. Incipient cataract left eye.

Fig. 4. Case 10467. N. Y. Eye and Ear Infirmary. Mature cataract right eye.

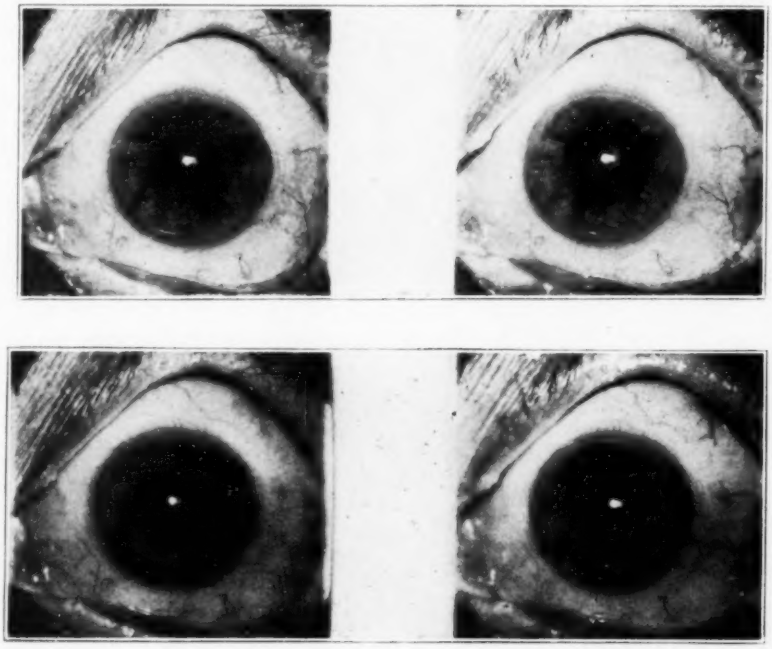
If careful refraction is done and fields, both central and peripheral, central color vision, light sense, and blind spots are studied, the cause of the changes in visual acuity can usually be found.

Figs. 3 and 4 demonstrate the value of photographs in recording and advanced cataractous changes.

We could do no better than to adopt the methods outlined by Bedell and in addition make comparative studies of the pathologic material by the usual methods and by the dark field method and ultramicroscope as used by Cattaneo.[3]

In conclusion I wish to emphasize the care which must be exercised in evaluating stereoscopic photographs as taken with the Drüner or Zeiss cameras, because of the many variable factors which enter into photography. The illumination, as it is an integral part of the Zeiss camera, gives more comparable photographs. The location of the anterior focus of the camera may be so placed as to emphasize any plane in the anterior segment of the eye, and a lens may appear to be clearer than it is, if the focus is not adjusted for the same depth.

There are many variable factors in developing, and especially in printing, as illustrated by Figs. 5 and 6, which are prints made from the same plate.



Figs. 5 and 6. Miss J. T. Left eye, October 8, 1928. Figures 5 and 6 were made from the same plate and show the difference due to printing.

This word of caution is not meant to detract from the great value of photographic records in clinic and private practice.

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DR. CLARENCE KING, Cincinnati, O.: Doctor Bedell rightly stresses the necessity of examining the lens by lateral illumination with the opthalmoscope, and particularly with the plane mirror at a meter's distance. An instrument which is extremely practical for all these purposes, is the simplified diaphragm lamp of Gullstrand. It can be used for focal or diffuse illumination, and is especially valuable for transillumination of the lens with the concave mirror of the ophthalmoscope, employing a strong plus lens. By this instrument, an intensely brilliant area of focal illumination is obtained on the retina, an illumination which serves to render even slight opacities of the lens apparent, on the principle that an opaque object is outlined more definitely when held in front of a sharply defined, intense light source than when it is placed before a large, diffuse illumination.

However, we must depend on the slit lamp to complete the observations made by the previous methods. The slit lamp is, of course, not only an instrument of research, but it is an invaluable aid to the clinician. It affords information as to the progress of the cataract. In senile cataract, it informs us, at least approximately, as to the amount of clear cortex which exists. We are enabled by it to recognize and differentiate complicated cataract with greater certainty than with any other method. Slit lamp studies are essential in the planning of an operation in secondary cataract, and are indispensable in the investigation of traumatic cataract. It is to be hoped that by means of the slit lamp, supplemented by photography, we shall be able to exactly determine the form and extent of opacities in senile cataract, so that subsequent changes, either of advancement or retrogression, may be noted. In this way, many of the mooted questions in regard to the spontaneous or induced changes in senile cataract may be approached scientifically.

Doctor Bedell has confined his remarks to cataract which has developed after the adult nucleus has been laid down. This involvement of the lens he has described in detail, and has illustrated beautifully. It seems worth while to call attention to a classification of senile cataract by Meesman which, tho it presents nothing new, is extremely practical. Omitting congenital changes, cataract in the adult can be conveniently classified into presenile and senile cataract, secondary cataract, traumatic, and complicated cataract. Meesman divides senile cataract into nuclear and cortical cataract. Nuclear cataract can occur as an isolated form or in combination with cortical cataract. Cortical cataract can be conveniently classified under two main headings. The first group comprises the opacities in the cortex due to the pathologic absorption of water and its consequences, and the second, the primary opacities due to the disintegration of lens substance. Cortical water absorption manifests itself

commonly as the well known "clear clefts," as lamellar separation, and as subcapsular vacuoles. The clefts may be due to the separation of the sutures of the lens by aqueous fluid or to the collection of water between the lamellae. The second type of water absorption lamellar separation appears as a system of fine, parallel, white lines, particularly marked in the nasal inferior quadrant of the deeper cortex. This phenomenon is not fully explained, but is attributed by Vogt to the separation of concentric lamellae of the lens by water. The third and last form of water absorption is the subcapsular vacuoles or, when extensive, the subcapsular vacuole surface of Vogt. The second main subdivision of cortical cataract includes the opacities due to the primary disintegration of the lens fibers themselves. The chief representative of this group are the cuneiform cataract, the senile point and dust like opacities, and particularly the posterior, saucer shaped cataract. This latter form of cataract is of great importance, as it causes decided reduction in vision, and because it must be differentiated from complicated cataract and from posterior, rosette shaped, traumatic cataract. Many who have observed with the slit lamp posterior saucer shaped cataract with but slight cortical involvment, must have been disconcerted, like myself, by the large amount of apparently clear, normal cortex present. Such a condition suggests the need, in extracting such a lens, of ripening methods, irrigation, or removal in capsule. It is well, however, to recall that the term senile nucleus of pre-slit lamp days, as used clinically, does not correspond to the senile nucleus as outlined by the characteristic band of discontinuity as observed with the slit lamp. The precise extent of sclerosis of the lens is not so easily determined by the slit lamp, and, of course, as age advances, it may extend almost to the capsule.

The investigations of Vogt have now definitely established the fact that uncomplicated senile cataract is not subcapsular in its inception. It begins in the deeper or middle layers of the cortex. It is significant that the complicated forms of cataract which are due to lentoexogenous influences have a subcapsular origin. These facts are the basis of the well known belief of Vogt, in which he is supported by many others, notably Elschnig, that senile cataract is simply a natural aging of the lens, similar to the greying of the hair or the changes in the bones, and that it is, therefore, not susceptible to therapeutic modification by nonoperative methods.

I hope that subsequent speakers will discuss a form of cataract which occurs in the adult and which is caused by tetany. This cataract has a typical appearance, and may be due to the disease or the removal of the parathyroid glands, or may be produced experimentally. It is also of interest to consider the possibility of latent tetany existing as a factor in the ordinary types of senile cataract. This entire subject is intimately bound up with the questions concerning the relation of the glands of internal secretion and lenticular opacities, so interestingly developed by Siegrist in his recent monograph on senile cataract.

DR. E. E. BLAAUW, Buffalo, N. Y.: We all owe our thanks to Dr. Bedell for his great enthusiasm in this subject.

I do not think the primary changes in the senile cataract is an elementary process. I think that has not been shown by the pictures. It

has been proven that many forms of cataract are due to changes in the internal secretions.

The initial changes in the lens may be due to senility of the tissues, and I think it is possible at a certain moment, when the cataract is just beginning, when it is in the initial stage, by rebuilding the whole organism to perhaps stop the progress. We will not be able to reduce opacities, but I want you to let go of the idea that there is nothing to be done with senile cataract. I think it has been beautifully demonstrated that we must not be so pessimistic, but that we must try if possible to influence the progress of the cataract.

DR. ARTHUR J. BEDELL, Albany, N. Y., (closing): Dr. Blaauw recalls that at a certain stage of cataract we have second sight but we know that sooner or later that subsides and the vision is lost.

Dr. Berens and Dr. King have shown their appreciation of the opportunities for study and record made possible by cataract photography. Unfortunately, it is impossible to show you these cases on the screen with the detail you will get in your office.

In closing, you will remember that I said in my paper in the Pre-session Volume:

"As yet no treatment has been discovered to restore clearness, or bring back to usefulness the opaque lens fiber. Any claim of this has not been substantiated by photographic proof. Visual function is not a guide as to the process transpiring within the capsule. The act of vision is a complicated mechanism, and the clarity of the lens is only one of the many factors involved."

THE OPERATIVE PROCEDURES AND METHOD OF HANDLING JUVENILE CATARACT.

WILLIAM H. WILDER, M.D.

CHICAGO, ILL.

THE LENS NUCLEUS.

For consideration of the method of their treatment, cataracts may be classed as hard and soft, by which is meant that hard cataract is one in which the lens nucleus has become sclerosed and hard, while in soft cataract the nucleus, altho present in the embryonic or early adult form, is soft.

The crystalline lens at the third month of fetal life is almost globular, being made up of the growth of elongated cells that have developed from the invaginated lens vesicle of the ectoderm. The formation of successive layers of cells from the vascular lenticular capsule exerts a pressure that flattens the structure from before backwards, until at birth the crystalline has assumed the biconvex lens form. Growth continues, and the development of superficial layers from the cells of the lens capsule compresses the layers in the center into zones of firmer, harder tissues, the nucleus.

The slitlamp has made it possible to differentiate the various layers of the lens structure by the difference in the refractive indices of the various zones of discontinuity. With the narrow beam and adjustable slit, Vogt has mapped out the different parts of the lens and has demonstrated the embryonic nucleus in the newborn and also in the fetus. Outside of this nucleus is another, the fetal nucleus, that, in its formation, corresponds to the time just before or at birth.

Outside of this are seen the reflecting surfaces that bound the larger senile nucleus, the Alterskern of Vogt, better called adult nucleus according to Harrison Butler, as it is seen in persons as young as the age of ten and has nothing to do with senility. Butler states: "In children this zone is not yet well differentiated, and probably the adult nucleus in childhood is the equivalent of the cortex in the older lens, a layer that has not appeared at

twelve. In the fully developed lens, and still more in the senile organ, the adult nucleus is sharply demarcated from the cortex and frequently by a well defined line of yellowish color."

This comes about by the growth of the cortical layers, and altho growth is retarded when adult life is reached, it nevertheless continues slowly until the end of life. Priestly Smith has shown that the senile lens is distinctly larger than that of young adult life.

But with this growth from the epithelial cells of the anterior capsule and the addition of successive external layers, there occurs a shrinking of the older central layers, probably from compression and the loss of moisture, and thus the harder, more resistant nucleus is formed, which, however, retains its transparency.

The slitlamp and corneal microscope should help in determining the character of a juvenile cataract, and such knowledge may be valuable in deciding on the best method for its removal. The slitlamp will show the relative size of the embryonic, fetal and adult nucleus, but does not give us such complete information as to its hardness or sclerosis, which is an important point for the operator if he wishes to perform discission. The existence of small vacuoles and small areas of degeneration thruout the nucleus might speak for a soft nucleus that would break up readily following a discission. A brownish coloration or a diffuse grayish haze, such as one frequently sees in senile lenses, would indicate a hard sclerosed nucleus that would resist absorption.

The study of the surfaces of the adult nucleus with the slitlamp may give some information as to its comparative age and density, thru the "relief" modeling observed on these surfaces. The suture lines and knob like protuberances, cameo like on the anterior surface and depressed, like an intaglio, on the posterior surface of the nucleus, have been extensively studied and pictured by Vogt and Lussi.

In a study of 628 cases of ages from 1 to 90 years, to determine the age at which the "relief" could be observed, they found it pronounced or slight in 193, or 30.7%. In ages before 10 years, 7 cases showed no relief. In ages to 20 years, only 1 case in 91 showed relief, and in ages to 30 years, of 207 examined only 14 showed relief, less than 1%.

From these observations it would seem that the phenomenon of relief is suggestive of advancing hardening of the nucleus.

However, the age of the patient will be our chief guide in

determining this point, and we can usually safely say that in the period of childhood and adolescence there will be no hard nucleus to complicate the operation. The slitlamp may give us more information than is obtained with the loup as to anterior or posterior polar cataracts, or membranous cataracts with iris adhesions, that might present distinct contraindications to treatment by discission.

Principle of Treatment by Discission.

The method of treatment of cataract by discission depends upon the fact, that if the capsule of the lens with its epithelial lining is cut or torn so that the aqueous can come into direct contact with the lens substance, the protoplasm of the lens fibers will swell up, become opaque and then gradually break down from fatty degeneration, so that they will be absorbed by the fluids of the eye. In this way after a single puncture of the capsule, the resulting traumatic cataract may be completely absorbed in a young child, leaving a clear black pupil.

It was undoubtedly suggested to the early operators who advocated it by the observation, that frequently after a simple traumatic cataract from a penetrating wound of the cornea or after an unsuccessful attempt at couching the lens, the cataract would disappear by absorption.

Altho it is suspected that the ancients may have made use of this method, Percival Potts is given credit for the discovery, in 1775, that if the capsule of the lens was freely lacerated, the cataract would be absorbed by the action of the aqueous.

Conradi, in 1797, observed after puncturing the capsule during an extraction which he was obliged to discontinue, that in eight weeks the pupil became quite clear. Later he limited operations on the capsule to soft or fluid cataracts. About the same time, Saunders believed that he was the first to discover by discission a new method of curing cataract in children, but he did not continue his work long enough to demonstrate that it is not suitable for senile cataract or those in which a large nucleus had formed.

Cases Suitable for Discission.

As has been intimated, discission is not the proper operation for cataracts in those above the age of twenty-five or thirty years, for the nucleus becomes so hard and resistant to the fluids, and in old persons is so large, that it will not be removed by absorption but must be extracted.

I have, however, used this method as a preliminary to linear extraction to be described later, in one case as old as 40 years finding a nucleus that could be extracted thru the keratome incision.

Nor is the method applicable to all cases of congenital or juvenile cataract. Obviously it would not be used for congenitally subluxated lenses that were cataractous, for the loosely attached lens would recede from the instrument or might be completely luxated.

In anterior polar cataract, either congenital or acquired, the circumstances of the case would help to decide whether discission, or this combined with linear extraction, should be practiced, or optical iridectomy performed.

It would not be the operation in certain cases of posterior polar cataract, particularly those associated with a postlenticular membrane and hyaloid artery remains. It is not suitable for shrunken or membranous lenses, either congenital or traumatic, in which the capsule is frequently tough or calcareous. It is the operation of choice in all other soft cataracts, whether of the congenital or acquired, nuclear or perinuclear varieties.

Operation of Discission.

At the first examination the pupils should be dilated, not only to study the character of the cataract, but also to note whether there are evidences of previous inflammation in the form of synechiae, that might complicate the operation by preventing mydriasis.

It is well also to inform some members of the family of the nature of the treatment, and that it may require two small operations and in certain cases, three or even four, before the final result of absorption of the cataract is obtained.

Neglect of this will often cause dissatisfaction and lack of confidence of people who are expecting immediate and brilliant results.

I agree with Treacher Collins, that usually it is best to wait until an infant is ten months old before discission, so that the anterior chamber will be larger and allow more room for the necessary manipulations. However, there will be exceptions to this rule.

In the preparation for the operation, the general condition of the patient should be attended to. In older children, the possi-

bility of focal infections from teeth, tonsils, or adenoids should receive consideration.

If there is tearing or discharge from the eye, the tear sac should be examined with a view to congenital obstruction of the tear duct.

Cultures from the conjunctival sac, on blood agar, should be made two or three days before operation. If pneumococcus streptococcus or staphylococcus aureus is found, operation should be deferred until treatment of the conjunctival sac with irrigation clears up the condition, and subsequent cultures give negative results. It is wise to insist on this, for discission is thought to be such a simple affair, that the usual preparatory antiseptic technic of the more pretentious operations is sometimes neglected, often to the misfortune of the patient and the sorrow of the operator.

The pupil should be widely dilated with atropin before the operation.

General anesthesia will be necessary for infants and young children, while local with 4% cocain will be sufficient for young adults and children old enough to cooperate.

The same careful antiseptic preparation of the conjunctival sac that is employed in senile extraction should be practiced on this.

The instruments to be used are eye speculum, fixation forceps and either a Bowman needle, a Knapp capsule knife needle or a Ziegler knife. Of these, I prefer the latter because of its cylindrical shaft, which allows it to glide in and out of the small corneal wound without loss of aqueous. One should see to it, however, that the blade is so constructed that it does not make a larger wound in the cornea than is blocked by the shaft, otherwise aqueous is lost in the manipulation and the operation made more uncertain. This is apt to happen with the Knapp knife, whose shaft is slightly conical, tapering to the blade. The small opening made by the blade is completely blocked by the tapering shaft so long as the instrument is pressed forward, but the slightest withdrawal of the instrument, as in cutting, results in loss of aqueous which may complicate the situation.

It is usually best to make the puncture at the limbus, just at the overhanging margin of the sclera, for at this point there is some vascularity of the cornea to prevent possible infection and also to aid in the rapid repair of the little wound. I cannot agree that it is just as wise to make the puncture in the clear cornea

or that the operator has any advantage in making it there. It is true there is less likelihood of touching the iris, but this is usually easily avoided. The blade of the instrument is passed thru the anterior chamber until the tip engages in the anterior capsule at a point on the pupil margin diametrically opposite to the point of entrance, when the instrument is rotated sufficiently to bring the cutting edge directly against the capsule. By slight pressure and slight withdrawal, a straight incision is made, usually horizontal, along one of the meridians and thru the anterior pole of the lens sinking slightly into the lens substance. Without withdrawing the instrument, the eye is slightly turned with the fixation forceps and the direction of the blade changed, so that a similar incision is made across the capsule thru the anterior pole and nearly at right angles to the first. The instrument should then take its original position and be quickly withdrawn, so that there will be no gush of aqueous thru the little corneal wound that might allow the margin of the iris to attach to it. The importance of having the pupil well dilated and of having a keen knife blade not more than 5 mm. long, whose perfectly cylindrical shaft will completely stop the opening made by the puncture, is obvious.

Atropin is instilled and the usual dressing applied only to the operated eye. Very young children would better have arm splints applied, to prevent them from meddling with or tearing off the dressing.

If the knife is not sharp, or the operation is not carefully performed the zonule may be torn and the lens partially luxated.

It is better at the first operation to content oneself with a rather small incision in the capsule, not knowing how much swelling of the lens substance or how much reaction will follow. Some eyes are more sensitive than others to lens substance, and if one were suspicious of this, a preliminary test with lens antigen should be made.

However, the incision should not be merely a scratch, but should make a rent thru the lens capsule and its cellular membrane, in order to have the desired effect. Without an opening thru the cellular layer, there would be no chance for the aqueous to come into contact with the lens fibers and to bring about the necessary swelling and absorption of them.

In some cases, particularly in very young subjects, the amount and rapidity of absorption of the lens after the first tentative discission of the lens capsule is surprising, and one is

gratified to observe two or three months after the operation that nearly all of the lens substance has disappeared and with very little reaction. Should a more extensive laceration of the capsule and lens be made at the first attempt, it may be followed by rapid swelling of the lens substance sufficient to cause considerable reaction, that may manifest itself by iritis with posterior synechiae, marked ciliary injection, and even hypertension of the eyeball.

The instillation of atropin, 1% solution, regularly after the operation, once a day, together with cold applications, may help to avert this condition, but once the reaction has started it is probably better to use hot applications for twenty minutes several times a day, together with dionin 5%, two or three times daily.

If the lens is greatly swollen, with its substance protruding into the anterior chamber, and the hypertension persists for several days in spite of the measures above mentioned, the lens substance, or part of it, should be evacuated by the linear method of operation presently to be described.

I have observed that the young eye is much more tolerant of hypertension than the adult eye, and it may be less likely to have its retina and optic nerve affected by this secondary glaucoma. Possibly this is because of its thinner and more elastic scleral coat. However, if the tension remains fairly high for several days, about +2, equivalent to about 40 to 50 mm., especially if accompanied by ciliary injection, and there is loose lens substance in the chamber, it would be wiser to evacuate part of it. In young persons, one will frequently get a more accurate impression of the tension with the fingers than with a tonometer.

My own experience is not in accord with the idea of treating cases of soft cataract by an incision thru the entire lens, the thru and thru operation advocated by the late Dr. Ziegler. Not only is this procedure likely to cause undue reaction in certain cases, but it may luxate the lens by tearing the zonule. Furthermore, there is an inevitable mixing of the lens substance and the vitreous, and it has seemed to me, that when this occurs the breaking down and absorption of the cataract is decidedly delayed, and there is greater likelihood of the formation of an after membranous cataract than if the posterior capsule remains intact until the lens substance is mostly absorbed.

Hence I prefer to perform two or even three less extensive operations to accomplish this, reserving the discission of the posterior capsule, if that should prove necessary, for the final

operation. In very young subjects particularly, the extra time required does not count for much, if a good result is obtained. It is well to wait for eight or even ten weeks before repeating the discission, if the study of the lens and its capsule shows that the discission was made and the lens is absorbing. In the older adolescent and young adult patients, the absorption is apt to be slower, and one should proceed with caution.

The treatment of complicated, anterior and posterior polar or unusual forms of soft cataracts will be considered later.

Linear Extraction.

In the event of its becoming necessary to remove the lens or part of it following a discission of the capsule for soft cataract, the preferable method of procedure is the socalled "linear extraction." The same kind of operation is applicable in traumatic cataract in subjects younger than twenty-five or thirty years, in whom the lens nucleus is probably still soft or very slightly sclerosed and therefore quite small.

The operation is done with a lance shaped keratome, fairly broad, so that one can vary the length of the incision, depending on the presence or absence of any sclerosed nucleus. The incision should be made a shade back of the opaque border of the superior sclerocorneal margin and not as far back as when making the keratome incision for a glaucoma iridectomy; the knife should be passed into the anterior chamber carefully so as to keep the line of the incision in the opaque sclerocorneal border. Obviously, one should avoid wounding the iris, and if it should appear from the shallowness of the chamber that this is likely, one need not hesitate to make the small incision with a narrow bladed cataract knife as is done in acute glaucoma.

If it is known that the lens is entirely soft, the length of the incision need not exceed five or six millimeters, but if there is any likelihood of the existence of a small nucleus, it should be made longer, eight or nine millimeters, so as to facilitate any necessary manipulations in the chamber.

Thru this small opening there will be ample room for the extrusion of the soft lens material by the insertion of a narrow grooved blunt spatula and the gentle stroking of the cornea with a cataract spoon from below upward. These may be supplemented by the use of a suction canula or a hollow irrigation spatula, which should be ready for the occasion. If in an older patient a small disc like lens nucleus should appear in the anter-

ior chamber and not be washed out by the irrigation, it may be necessary to introduce a narrow wire loop to effect its removal. It is not necessary to be too diligent or fussy about removing every vestige of the lens substance, unless it be a lens nucleus, for these particles will be absorbed. It is needless to say that all this is to be done in the strictest aseptic manner and with the utmost gentleness, to avoid a possible rupture of the zonule or the wounding or prolapse of the iris. The skilful use of the grooved spatula, inserted into the wound to hold back the iris and along which the lens material will escape, will do much to prevent these accidents.

Following the operation, atropin should be instilled and the customary dressing applied, which can be changed daily. If the incision has been properly made, because of its shortness and the accuracy of apposition of the lips of the wound, there is little danger of subsequent gaping and prolapse of the iris, so that it is justly regarded as one of the safest of the cataract operations.

Discission of the Posterior Capsule.

This final operation may be necessary if the pupil does not show a clear area in the center. As a result of imperfect absorption of the lens, together with the reaction following the preceding operations and the folding and crinkling of the capsule, there may remain a more or less dense membrane stretched across the pupil that will prevent good vision unless a clear opening can be made thru it.

The same condition may exist after a traumatic cataract from a perforating wound of the cornea, in which case similar treatment would be applicable. It is necessary to make a central opening thru the membrane, but the method of accomplishing it may vary to meet the needs of each case.

The pupil should be dilated as widely as possible. The same careful precautions and aseptic technic should be observed as in any other operation in which the eyeball is entered by an instrument.

If the membrane is thin and adhesions absent or not numerous, the discission can easily be made with a Ziegler knife or with a Knapp knife needle. The instrument should be inserted at the limbus and care should be taken to make the cut thru the membrane in such a way that the resulting opening will be opposite the center of the cornea. Ingenuity can be used in taking

advantage of thinner spots that lie in the field of attack, and possibly enlarging or combining them.

If the membrane is denser and adhesions are present, I much prefer to divide it with two instruments in the following manner:

Good focal illumination should be used so that all the finer details of the membrane and adhesions can be plainly seen. With fixation forceps, an assistant holds the eyeball, grasping it below the cornea in the usual manner. He is to turn the globe toward the temple or toward the nose as the operator directs. If the right eye is the field of operation, the surgeon holds a Knapp needle in his left had and a Ziegler knife in the right. Directing the eye to be turned slightly to the right, so that there is room to work over the nose, he introduces the blade of the instrument in his left hand into the anterior chamber at the nasal limbus, and has its point perforate the pupil membrane at a point toward the temporal side of the pupil, holding it there. The eye is then turned to the center by the assistant, and the Ziegler knife in the right hand is introduced at the temporal limbus and its blade inserted into the membrane a little to the nasal side of the center 3 or 4 mm. from the point of insertion of the first instrument. The blades cross, and with proper care their edges can be brought together so as to act like the blades of scissors in dividing the membrane between the two punctures. This is accomplished by approximating the handles, and the rent in the membrane thus cut is made wider by separating the points of the instruments. The advantage of this method is that there is less pull on the membrane, and therefore on the ciliary body, than if one knife alone is used, for each pulls against the other, and consequently there is less likelihood of exciting unfavorable reaction.

Complicated After Cataracts.

Congenital membranous cataract is sometimes seen, which has resulted from an intraocular inflammation in fetal life that has caused partial or complete absorption of the lens substance. Usually in such cases, as in traumatic cases, there is the complication of marked iris adhesions to the membrane from the previous inflammation. There are frequently calcareous deposits, not only on the capsule, but thruout the remaining lens mass. I once removed a shriveled lens mass of this kind that seemed like pure chalk.

Such cases are not amenable to treatment by discission. The chamber should be opened as in the linear operation and an attempt made to cut thru the membrane with fine de Wecker scissors, one blade of which is made sharp pointed for the purpose of perforating the membrane. After this, each half is seized with flat, toothed forceps and dragged into the wound. If it is delivered in this way, well and good; if not, a piece of it can be severed with the scissors.

I have on several occasions used this procedure: In making the keratome incision, the point of the knife is pushed on thru the membranous cataract at the lower pupil margin, and an attempt made to enlarge the opening and sever some of the lower iris adhesions by side to side movements with the point of the knife. A rather long and somewhat narrow keratome is best for this purpose. Then, with a small sharp Tyrrell hook, the upper lip of the cut in the membrane is seized and the hook rotated one half turn with the fingers to secure a firmer hold. It is then drawn slowly up thru the wound and, if one is successful, a piece of it can be cut off with scissors, leaving a hole in the pupil area.

Unless it should seem loosely attached and yield readily, one should not attempt complete evulsion of the membrane. Such cases are desperate, and frequently after operating the resulting reaction fills up with exudate the opening that has been made, and the eye is lost.

Anterior Polar Cataract.

This may be congenital or acquired. In either case, the spot is frequently so small that vision is not much interfered with and operation is not called for. In some cases, however, the lens in the region of the polar nodule begins to degenerate and operative intereference is required. In such cases, optical iridectomy, advocated by some, does not seem to be the proper thing. Sooner or later the partial cataract will advance and if it has already invaded the cortex and the patient is under twenty-five, the lens should be removed by discission and the linear method. In these cases as we know, the nodule is beneath the capsule and is made up of proliferated capsular epithelium that is almost fibrous in consistence. It sometimes forms a boss or knob on the anterior capsule that protrudes forward like a cone, the socalled pyramidal cataract.

In operating on such a case, one should consider the firm

fibrous nature of the protruberance. In making the discission, one should avoid cutting thru the nodule, but cut the capsule below it. Later when the lens swells and the eye is opened with the linear incision, the nodule should be picked off with capsule forceps to prevent it falling to the bottom of the anterior chamber, where it would fail to be absorbed and would cause irritation if not removed.

Posterior Polar Cataract.

This is usually seen as a congenital spot on the posterior capsule, and the slitlamp reveals it associated with remains of the hyaloid vessels. In more marked cases, greyish strands in the vitreous connected with a membranous growth on the back of the lens, together with persistent hyaloid vessels, suggest glioma of the retina, for which such eyes have sometimes been removed. At first such lenses may be clear, but undergo cataractous degeneration later. If by discission such a lens is absorbed, there will still remain a dense greyish membrane connected with the vitreous, for which nothing satisfactory can be done. Naturally, for such conditions no operation should be attempted.

Summary.

For the purpose of treatment cataracts are classified as hard and soft, depending upon the condition of the nucleus of the lens.

The lens nucleus as seen with the slitlamp microscope. This instrument will enable us to estimate the size of the nucleus, but not so well the degree of sclerosis or hardness of it, a point of information of value to the operator.

Relief phenomenon of the anterior and posterior surfaces of the nucleus may be an aid. Age of patient most important guide. Under twenty-five years, the nucleus is probably soft and amenable to treatment by discission and absorption; over that age, the nucleus begins to harden and will require different treatment.

Principle of treatment of soft cataract by discission and cases suitable for it. Description of preparations and technic of operation and results obtained by it.

Supplemental operations necessary in certain cases to remove swollen lens substance. Technic of operation of linear extraction and indications for its use.

Subsequent discission of the posterior capsule. Double needle operation valuable if membrane is tough or dense. Complicated membranous cataracts congenital and traumatic. Anterior and posterior polar cataracts.

DISCUSSION.

DR. ALLEN GREENWOOD, Boston, Mass.: I have one or two suggestions which I will take up in five paragraphs.

First: As to the age of the patient, that depends somewhat on the type of cataract, and somewhat on the amount of vision that the child has. They are not all complete cataracts, but I prefer to operate fairly early if the child is blind. If the child can see to get around and play, I wait longer before operating.

Second: As to the instrument, I prefer at the present moment a Hay-Ziegler knife needle. In conjunction with the instrument, I would like to speak of its application. I join Dr. Wilder in deploring any entrance into the vitreous body with a knife at the time of operation. I do not believe it is necessary. I do, however, believe that it is essential that the incision in the capsule shall be large enough. If you make a fine puncture of the capsule, the lens substance has no chance to come into the anterior chamber if it swells. Personally, I have never seen a case of glaucoma following a free cut of the capsule that did not involve an extensive stirring up of the lens. I let the aqueous take care of the lens substance, which it will do if the capsule is properly open.

Another point is the place of entrance of the knife-needle. Personally, I do not like to go thru the cornea at all. I find no trouble in making my discission by picking up a conjunctival flap with the point of the knife-needle, and then puncturing thru as far forward as I can and still have a conjunctival covering for the wound. When I withdraw the knife, there is occasionally a tiny lifting of the conjunctiva, but I see no aqueous escape in such cases.

In traumatic cataract, which is considered but little here, the question of the lens producing swelling in the capsule is to be considered. This I have not had in any of my congenital cases, but a linear extraction is the proper method for removal.

I agree with Dr. Wilder, that when cortical material is absorbed, after cataract occasionally forms, necessitating a needling. I have had to do a few of these cases, for with a wide opening in the anterior capsule, the posterior capsule remains transparent, but in a few cases I have had to needle, where the absorption has not been good or where the anterior capsule has partly come together and there is a secondary cataract made up of capsule and lens material. A Hay-Ziegler knife is very good for this. Dr. Wheeler's discission with a very narrow Graefe knife is another good method. If occasionally I find calcareous material, as Dr. Wilder has suggested, I have used a Terrill hook to draw the material out and cut it off. These cases of after cataract that I have operated on have been mostly cases that have had one or two discissions before they came under my care. We all dislike to take patients who have been to

other men, but sometimes it is necessary to do so, and if they have been operated once or twice by discission, we sometimes get rather bad results, especially where the discission has not been thoroly done. I have a boy patient who, at the age of five, had single discission done on each eye, with complete absorption of the lens and two black pupils. These discissions were done with a long vertical opening in the capsule and a cut across.

Dr. John O. McReynolds, Dallas, Tex.: I wonder if we appreciate and instruct the people in general to fully recognize the importance of this subject of juvenile cataract. In the extraction of senile cataract, our only purpose is to add a few pleasing visions of the fading landscape of life to an old man tottering on the verge of the grave, but in juvenile cataract, the problem involves the entire span of life from its dawn until the sun goes down forever. The responsibility, therefore, is very much greater with reference to juvenile cataract than with reference to senile cataract.

The term "needling" is sometimes misleading, because it may suggest such a slight thing, that we may not always advise parents sufficiently of the gravity of the procedure. Personally, I feel there is just as much risk in needling as in extraction.

I am in thoro accord with everything that Dr. Wilder and Dr. Greenwood have said. There are two definite problems to be considered in the operative management of juvenile cataract. First, how can we secure the maximum amount of absorption; second, what are we going to do with that portion of the cataract which refuses to undergo absorption?

With reference to the first question, we must remember that practically all of the efficient absorption takes place in the anterior chamber, and not in the vitreous chamber. Therefore, any procedure which means cutting the lens entirely thru into the vitreous itself, exposing the lens substance to the vitreous instead of to the aqueous, fails to accomplish what we wish to do, because it is the aqueous which best helps to break down the lens fibers, and it is the iris that carries thru to completion the process of absorption. We are all familiar with how impotent the vitreous chamber is to take care of any kind of foreign material, such as blood and exudates. It is only in the anterior chamber that absorption rapidly takes place. Therefore, the point made by Dr. Wilder and Dr. Greenwood, that we should make a sufficient incision thru the anterior capsule to allow free access of the aqueous to the lens, is most important.

The essential thing then would be to place the eye in the best condition possible to avoid inflammatory reaction. We cannot emphasize too much the necessity of getting thoro dilatation of the pupil before we begin. There should also be a careful study of the case with both the slit lamp and corneal microscope and stereoscopic photography, and by an investigation of the patient's systemic condition. Many of these patients we do not feel safe in operating on until we have determined their true condition. Sometimes they are poorly nourished and not able to walk, and there is lack of development, not only of the lens and other parts of the eye, but of the entire body. They are poor patients. Why not wait

until you have as good a chance as possible before undertaking any kind of operation?

The dilatation of the pupil should be thoro, even if it takes two or three days to accomplish it. Frequently, it is advisable to inject underneath the conjunctiva a weak solution of cocain and adrenalin chlorid and atropin, so as to get mydriasis to the maximum degree. Then, when you have secured all the absorption you can, what are you going to do? This will depend on the condition of the anterior portion of the eye. If the eye has been the seat of much inflammation and the whole ciliary region is covered with organized exudate, you do not get good retraction of your capsule when you make an incision thru it. Then you must remove a portion of the membrane before you can get satisfactory results. Make a corneal incision, draw out a portion of the membrane with forceps and cut it off, thus making a new pupil corresponding to the location of the obstructing membrane.

I feel this is one of the most important subjects that may confront the ophthalmic surgeon, and we should not undertake this operation without giving full information to the parents of the child, and without appreciating to the fullest extent our own responsibility in the outcome.

Dr. W. A. Fisher, Chicago, Ill: Dr. Wilder has presented this subject so thoroly that there seems little to add, and the method he has advocated will probably meet with the approval of the majority of ophthalmic surgeons; but there are disadvantages in repeated needlings, together with a long drawn out treatment. There are times when it seems advisable to make an effort to finish the work in one operation and shorten the treatment. In such a case, it is well to insert between the lids an ointment of 5 per cent cocain and 5 per cent euphthalmin every forty-five minutes, until the pupil is fully dilated. The eye is then anesthetized, an incision made with a cataract knife, and conjunctival suture inserted, but left untied. After capsulotomy by forceps, the lens is removed in the usual manner, the suture tied, and a peripheral iridectomy made near the puncture or counterpuncture, and eserin ointment, one-half of 1 per cent, instilled. The wound is usually closed the second day after operation when atropin ointment, one-quarter of 1 per cent is used.

Such a procedure, as well as a needling, should be followed by milk or blood injections, as given by many operators where a complication follows a cataract operation by any method, and a discission or linear extraction could well be considered a complication.

Dr. E. E. Blaauw, Buffalo, N. Y.: Two years ago I talked before this body on lenses, and I think I ought to say something about this subject.

First of all, I wonder that Dr. Wilder does not say any more about iridectomy, and about the other sections of the lens.

According to the United States Census of 1917, there were 9,242 blind from all causes in the United States; 3,217 blind from cataract, operated and unoperated; and up to the 19th year (juvenile cataract), 221 blind. It, therefore, is a grave condition and we must work on it assiduously.

Before the Englishmen knew anything about the slit lamp, and before there was any mention of it in the British Journal of Ophthalmology, in 1921-23, the term "adult nucleus" was known in this country.

If we have a child with a complete cataract on both sides, we should operate as soon as possible, because if we keep it shut, not only the eye will not develop, but the brain also, because it seems that the entrance of light has its effect upon the formation of the brain, and we often see a case, where even if there is no light reaction of the pupil, and even if the child does not mind the light, when the eye is opened afterwards the child is much brighter mentally.

Of course I have a slit lamp, but the practical thing is just the same I do not know any more than I did before I got the slit lamp. It depends upon the size of the opacity of the lens whether we operate or not, but if we do operate we have to learn to begin with a small incision. I say if you make a large incision you have to be very careful. The same way with subluxation of the nucleus in the lens.

Fifteen years ago, I did needling, and it took two months before I got the pupil. When I got at the nucleus, it took one month. But we should operate as little as possible.

I like the operation of Baer. He goes into the limbus and makes an opening a little bit longer than wide, and if the nucleus is further out, he makes a little bit longer incision, and he extracts the nucleus just as if it were an adult senile cataract.

Dr. Conrad Berens, New York City, N. Y.: To have heard such a stimulating discussion of the question of the management of juvenile cataract makes one naturally hesitant to suggest in the slightest way that his experience has differed from that which has been outlined. However, in the matter of Ziegler's complete discission of the lens, my experience and that of Dr. Johnson and Dr. Losey has not been similar to that of Dr. Wilder. Ziegler's (Jour. of the Amer. Med. Assoc., 1921, vol. LXXVII, p. 1100. Amer. Jour. Ophthal., 1922, vol. V, p. 381. Trans. Ophthalmological Soc., U. K., 1925, vol XLV, p. 154) exact method has not been followed in all the operations which have been done. Many of the clinic cases were performed with a preliminary iridectomy, which was first suggested and practiced by Albrecht von Graefe, and was again proposed by Meyer and Montméja (Meyer, E. and Montméja, A. de, Ophthalmic Surgery. H. Lauwereyns, Paris, 1871) in 1871. These surgeons performed iridectomy with anterior capsulotomy, and more recently, Bell (Bell, G. H., Operation for Juvenile Cataract in Two Stages, Amer. Jour. of Ophthal., 1926, vol. IX) has suggested combining iridectomy with Ziegler's complete discission of the lens. Bell reported 100 cases operated on by this method. In no case of his series upon which I operated or followed the after treatment, was it necessary to open the anterior chamber to express or wash out the soft lens matter. Furthermore, the reaction was very slight in practically all of the cases, and increase of tension, if any was noted, was much less than one usually sees after anterior capsulotomy.

In private practice, it has not always seemed necessary or wise to perform iridectomy, and it became necessary to wash out the soft lens matter in only one case. This patient had marked hypersensitiveness to

lens protein, and was seen by Dr. Verhoeff in consultation. The patient's hypersensitiveness to lens protein was recognized before operation, and he had been desensitized.

The method I prefer is to dilate the pupil *ad maximum* with atropin fortified by adrenalin—the technic recently advocated by Dascalopoulos in operation for traumatic cataract (Dascalopoulos, N. The Use of Adrenalin in the Operation of Traumatic Cataract. Annal d'Ocul. 1928, vol. CLXV, p. 251). The conjunctiva is nicked with scissors 6 mm. from the limbus, and a well curved Ziegler knife needle is then passed subconjunctivally and thrust thru the sclera ½ mm. from the limbus. The lens is cut thru from below upward with a sawing motion, a single straight incision in most cases having been found less difficult to make than the typical Ziegler incision. This has been previously advocated by Parker. Our experience differs from that of Ziegler in two respects: first, that it is never necessary to cut the secondary membrane (for several patients have required secondary operation); second, that the corneal route is to be preferred to the subconjunctival method of approach. The operation as described has been performed in patients under 25 years with congenital, juvenile and traumatic cataracts, and in high myopia. The advantages of complete discission of the lens appear to be:

1. Fewer operations required to obtain the same result.
2. Less reaction than was usually noted after anterior capsulotomy.
3. Secondary increase in tension less frequently encountered.
4. Danger of infection less, for the number of operative procedures is usually reduced.

Dr. Meyer Wiener, St. Louis: I hesitate to take issue with so eminent an authority, and a man for whom I have such high respect as Dr. Wilder and some of the men who have followed, but my experience of nearly twenty years with the Ziegler method, I feel, justifies my defense of this method and the extolling of its virtues. My experience has not coincided with that of Dr. Wilder and the other gentlemen. In the first place, something was mentioned about the vitreous in the absorption of lens material. I do not think that Dr. Ziegler ever advanced the idea that the vitreous was a factor in absorbing lens material. The aqueous penetrates thru the opening we make into the capsule into vitreous and swells up the lens, and it absorbs thru the aqueous, even tho one-fourth or one-half of the lens may fall back into the vitreous body. This has been proven by experiments made by Dr. B. Y. Alvis, my associate, upon animals' eyes, which will be published some time before long.

I think a lot of people condemn the Ziegler method because they do not follow out the exact method as advocated by Dr. Ziegler. In the first place, Dr. Ziegler laid emphasis on the necessity for making his puncture within the cornea. You cannot get a fulcrum for leverage unless you go within the corneal margin, and you cannot produce the proper sawing motion to get a cut without pulling on the zonule. In our experience, we have very seldom had to do a discission the second time when we have performed the Ziegler operation, making a bell shaped opening. There is no pulling on the zonule if the needle is sharp, and if the incision is made within the corneal margin, if you use the sawing motion, a second discission is not necessary.

As regards the age, I do not think there should be any limit. We operate on juvenile cataract any age they come to us, and we have operated as young as two months. There are some cases where we cannot obtain dilatation of the pupil. A lot of these cases are the result of intrauterine trouble. Very often, we have complete adhesion of the sphincter to the lens. I believe the Ziegler method of discission can be used in these, by making a nick in the iris both below and above the artificial margin of the pupil. I can see no objection to this method.

Dr. William H. Wilder, Chicago, Ill. (closing): I want to thank the members who complimented me by discussing this paper. Of course there always will be differences of opinion as to methods of operation, and it is so difficult to get accurate information and to deduce perfectly valid and logical conclusions from statistics on operations. I think I will meet with a good deal of accord in saying that usually a man should select that operation which he can do best. What might serve one man well might not serve another so well, so it is a good principle in operating, for a man to do that operation in which he feels the most confidence and with which his experience shows he obtains the best results. I have no criticism of the Ziegler operation, except that it seemed to me from the experience I had with it that there was delay in the absorption of cortical material of the lens.

With the thru-and-thru operation, you do take a chance of rapid swelling of the lens, and if later on it becomes necessary to open the eye by linear incision, you have the decided risk of loss of vitreous because of the presence of vitreous in the anterior chamber. So I prefer to move a little more slowly, if necessary making the operation of discission two or three times, and reserving for the final operation the dividing of the capsule and throwing the two chambers into one. I think on the whole that is considerably safer. I feel that the question of time should not enter into the operation. I quite agree, that it is well to defer the operation of discission up to a period when the eye is formed and there is a little deeper anterior chamber in which to carry on manipulations. On the whole, I prefer to carry on the treatment in this way, not stressing the matter of time at all. What is a year or two to these little subjects if we get a good result. It is better in any operation of this kind to make it as safe as we can.

There are one or two little points I would like to emphasize in closing the discussion. One is the question of the final operation, final discission; and the other the division of the membranous cataracts that we all see in which, because of more reaction than usual, there remains a rather dense membrane with adhesions of the iris to it and to the posterior capsule. Most of the lens substance has been absorbed, and there still remains this membrane across the pupil. In these cases, what operation will you do? If you can with a sharp Ziegler knife make an opening thru the membrane without too much dragging on the ciliary body, that is admirable; but with many of them, the adhesions are so firm that it is difficult to make the divison without a considerable pull on the ciliary and consequent irritation.

In these cases, I have had the best results with the use of a double needle, I think originally proposed by Bowman, and advocated strongly by Risley of Philadelphia. The technic of the method is described in the paper.

I quite agree with Dr. McReynolds' suggestion of introducing a small Tyrrell hook so as to snip out a small pinhole about the center of the cornea. A somewhat similar procedure that has given me some good results is considered in the paper.

CERTAIN DEBATABLE STEPS IN THE OPERATION FOR SENILE CATARACT.

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The *maturity* of a *cataract* is no longer waited for before operating, and an operation is now done as soon as vision is so far reduced as to interfere with the patient's livelihood or his ability to read. Some operators proceed at any stage of cataract formation, so long as the patient has passed his sixtieth year. The depth of the anterior chamber is for me the most important criterion, as the anterior chamber must be deep enough to permit a proper section, and we must always remember that the section is the most important step in the cataract operation. Clear subcapsular cortex is not a contraindication, if the lens can be extracted in its capsule, or an opening made in the anterior capsule large enough to allow access of aqueous to the retained cortex. The present-day examination with the powerful light, as is used in the slitlamp, permits us to determine in many cases if the posterior cortex be clear, and if a posterior cortical opacity is present, for in the latter case the cortex will be readily separated from the posterior capsule, and emerge with the nucleus. There is one form of whitish-gray cataract with thin, dense capsule, or one where the superficial cortex presents a peculiar asbestos like sheen, in which the blunt capsule forceps takes hold with difficulty, as it tends to slip off or is apt to rupture the capsule if the intracapsular operation is attempted. In the sclerosed type of cataract and in the nuclear cataract with posterior cortical opacity, the cataract is usually easily subluxated and extracted in the capsule, or in the way of a proper capsulectomy, the lens contents come away in a single mass. The type of cataract which should not be attempted lightly without full knowledge of its complications is the nuclear opacity in myopic eyes with a clear posterior cortex. In this, the cortex is necessarily retained, and complications due to the various forms of after cataract may ensue; then, under the best of circum-

stances, a discission becomes necessary, which is often more difficult than the primary operation. These cases do not lend themselves to the intracapsular operation, owing to the myopic structure of the eye.

In order to avoid the difficulties of retained cortex and its complications, operations to hasten the maturity of a cataract have been practised. These consist in an iridectomy, sometimes combined with massage of the lens thru the cornea, or direct massage of the lens capsule. So little is obtained by these, that they have been abandoned by most operators of experience. The discission of Homer Smith, followed by extraction after 12 hours, has not met with favor, as extraction on an inflamed eye is not good surgery.

Preliminary iridectomy is performed by many operators as a routine measure, and is regarded as a safety maneuver. It has the disadvantage of adding another operation to the cataract extraction, and interferes with the formation of a suitable conjunctival flap—a very important part in the corneal section. I cannot see that it has any advantages except in the slowly maturing nuclear cataracts, where additional sight for a time is often obtained, and in certain complicated cataracts where there is fear of iridocyclitis.

The question of iridectomy at the time of the cataract operation has long been a debatable one in ophthalmic gatherings. I practice an iridectomy as a routine, unless the operation is a cosmetic one in a young individual, because the iridectomy facilitates the intracapsular operation with the capsule forceps if that operation is determined upon, and in capsulectomy cases, the cortex is more easily extracted and iris prolapse is greatly reduced. Its objections are the tendency of the capsule in capsulectomy cases to be caught in and adhere to the corneal section—a very grave condition which is apt to be followed by glaucoma with all of its dangers. After the iridectomy, I immediately replace the columns of the iris coloboma with care. On the Continent and in England, the practice to operate with a peripheric buttonhole iridectomy is now general. It is a procedure which should carry the name of Chandler-Pflüger-Hess. I do not practice it, as it is not suitable for the intracapsular operation. In capsulotomy operations, it is undoubtedly of advantage. The round pupil is optically of advantage, and by this maneuver the danger of iris prolapse is reduced from 8-10% to 1%.

The most important difference in the cataract technic consist in the ways of dealing with the lens capsule. Thus the lens capsule can be opened by a cutting instrument, a cystotome, or a piece of the anterior capsule can be removed by a capsule forceps, or finally the capsule can be extracted in toto together with the cataract, the socalled intracapsular operation. It is my belief that the ophthalmic surgeon should be familiar with all of these procedures, because there are definite indications which call for each one. An old method of opening the capsular bag has recently been revived, in which the capsule is cut by the knife in making the corneal section. This procedure was abandoned by H. Knapp because the opening in the capsule was insufficient in many cases, and it made an exact execution of the corneal section more difficult.

The opening in the capsule in cataract operations was formerly always done with a cystotome. Various methods of using this instrument were devised, and while they theoretically seemed promising, actually the result was an extensive and promiscuous laceration. The opening in the lens bag is often unpremeditated in extent, as it tears along the lines of least resistance, and then this opening is enlarged by the issuing lens matter. A peripheric incision of the lens capsule was consistently used by H. Knapp, as this gave the easiest exit to the lens. Elschnig objected to this on account of the danger of injury to the suspensory ligament, particularly in restless patients. A vertical incision in addition was practiced for a time by H. Knapp, but was abandoned because after the vertical incision had healed, it formed a thickened capsular band which made the subsequent discission more difficult. Multiple incisions of the capsule with the cystotome often lead to a permanent opening of varying size, but a tag of capsule is apt to heal in the corneal section.

After opening the capsule with a cystotome, a discission has to be done in a large percentage of cases. Some operators do this systematically at an early stage after extraction; others wait for a later period, if the visual results are not satisfactory.

There is no question that the cystotome is the easiest instrument to use in opening the capsule, and it should therefore be the one with which the beginner starts, just as it should be used in complicated cataracts.

The capsule forceps was a later invention, and after a time, has been generally adopted. Numerous models which do not differ very much figure in the instrument catalogs. In applying

these, greater pressure has to be used than in the case of the cystotome, which may lead to a dislocation of the lens or prolapse of the vitreous in unpractised hands. The resulting opening naturally depends upon the thickness of the capsule. As a rule, if a toothed instrument is used, a tear is made in the capsule, and a piece of capsule is removed which varies in extent, but its exact size cannot be gauged. This leads many operators to make an additional incision with the cystotome (Weeks). In thin capsules, the result of the capsule forceps does not differ from promiscuous laceration. In thickened capsules, the entire lens and its contents can be grasped and pulled out. The use of the capsule forceps undoubtedly reduces the frequency of secondary cataract, altho it does not entirely eliminate it, as a secondary cataract may form from a wrinkling of the posterior capsule, often one or two years after the first operation.

The capsule forceps which I favor are the blunt capsule forceps of Kalt, which have a cupshaped form which grasps the capsule and tends to tear it, not at the point of application, but at the equator of the cataract; thereby, a very large piece of the capsule is removed. In many cases, however, before this rupture takes place, the lens becomes dislocated. This suggested to me the method of intracapsular extration which I now practice.

The advantages of the intracapsular operation are admitted. The early operation, the freedom from iritis, the avoidance of a secondary operation, and of the complications due to retained cortex and of capsular adhesions with glaucoma are so well understood, that they need not be mentioned in detail.

A number of methods for removing the lens in the capsule have been devised, but from its greater danger of vitreous loss with the resulting complications in a large percentage of cases, it can be said that up to the present time an ideal method has not been established.

All intracapsular methods now in vogue start with the dislocation of the lens, and after the lens is dislocated, the extraction is simple. According to the Smith method, this dislocation is accomplished by external pressure. This, in my opinion, is too great to be compatible with safety for the eye. The second method is the dislocation brought about by the capsule forceps. This is the safest method, but only succeeds in dislocating the lens in a proportion of cases, as the capsule tears. Finally, the dislocation has been effected by the vacuum cup. I have no personal experience with this method. It seems to me, however,

that the mechanism of the pump is complicated, and that in case of accident, the suction cannot be readily controlled or arrested as easily as when the forceps is used.

The intracapsular operation should of course be practiced only in selected cases: in patients who are amenable, where there is no complication on the part of the vitreous, where the eyes do not bulge, and the eyes are not myopic.

The dislocation with the capsule forceps, which is the method I advocate, consists in grasping the capsule in its lower third. The grasp of the forceps should be firm enough to hold the capsule, but not necessarily hard enough to tear it. The forceps are then moved from side to side, or up and down, putting the lens capsule below on the stretch. Then, with the blunt strabismus hook, external pressure is made at the lower limbus directly upon the suspensory ligament, and if the capsule is not unduly elastic, the cataract readily dislocates below, which will be noted by the lower margin of the lens coming up and remaining in the pupillary area. The subluxation is practiced below, because the vitreous is there protected by the intact corneal limbus. Vitreous is therefore much less frequently lost than when the luxation is above.

The capsule forceps is then promptly released, as subluxation has been affected. The section is enlarged if there is any doubt about its size. Then combining pressure with the hook below and the spoon against the posterior lip of the incision, the cataract is made to tumble; in other words, the lower margin comes forward, and the cornea is tucked under behind the presenting lens. During this procedure, very little pressure is required, and as soon as the equator of the lens presents, the direction of the pressure is changed to upward, and the lens is finally extracted by separating the adhesion of the superior part of the suspensory ligament.

The reason for selecting the lower third of the capsule is to dislocate the lens below, which is furthest away from the line of section where the vitreous is most exposed. Then, if the capsule ruptures, the resulting opening is just in the pupillary area. The method of grasping the cataract with the forceps above or at its center (Stanculeanou-Török) and then, after dislocating, pulling the cataract out by traction with the capsule forceps, is objectionable as in it the capsule more frequently ruptures, and we then have the complication of a subluxated and ruptured capsule to deal with, which means the subsequent ex-

traction of the capsule, which is not easy, and there is a greater percentage of vitreous loss.[1]

If the suspensory ligament below is elastic and adherent, and if moderate manipulation does not succeed in subluxating the cataract, the blunt forceps should be released and the procedure abandoned for the toothed forceps or cystotome. It is not safe to exert too much traction on the ciliary body.

An unusual complication is the subluxation laterally or above, especially from an untoward movement on the patient's part. The further steps then have to be very carefully followed out, as vitreous is apt to present. After the extraction of the lens in the capsule, after a few minutes delay, the iris columns are replaced and the conjunctival flap straightened out.

The results of this method of operating, I think, justify its adoption in selected cases. If the capsule forceps does not dislocate the cataract, the capsule ruptures and a large piece of capsule is removed and the usual operation can be proceded with. This dislocation takes place so easily in many cases that it seems justified to give the patient the advantage of an intracapsular operation; if it fails, nothing is lost, and at any step the procedure can be changed according to the exigencies of the case.

I feel that the intracapsular method is not an operation with which one should begin his operative experience, and the apprenticeship should be served by following out other and simpler methods. The operator must be prepared to deal with complications such as vitreous loss either before or after the delivery of the lens.

The visual results which I obtained with the intracapsular method in 200 successive cases were published in the Archives of Ophthalmology[2] in 1915 and 1921. The effect of loss of vitreous has also been carefully studied, and while I in no way wish to minimize this complication, as the prolapse of vitreous takes place at the conclusion of the operation it is not serious. That the good visual results are permanent, and that there is no such thing as late vitreous degeneration were shown in my paper read before the International Congress in London in 1925,[3] in which it was possible to follow up and reexamine 85 out of 200 intracapsular extractions after ten to fifteen years. The great freedom from glaucoma, detachment of the retina and severe iridocyclitis in this series was noteworthy.

Let us now turn to the capsulotomy cases and consider the

complications which are primarily due to the lens capsule. After cataract depends on the method of capsulotomy employed and on cortical matter remaining at the operation. An opaque secondary membrane is unlikely if there is no retained lens matter, no proliferation of the capsule nor iritis. To determine the incidence of the after cataract and its relation to methods of dealing with the capsule at the extraction, led me to study 200 successive capsulotomy operations from my private records, which I presented in a paper before the Section on Ophthalmology at the A. M. A. meeting in Minneapolis this year. The operations were combined extractions for senile cataract, performed before the end of 1926. The results were given in the following tables:

		Secondary Operations.		
	Extractions	Discision	de Wecker	Extraction
Blunt capsule forceps	87	4	2	
Toothed forceps	76	5	3	
Toothed capsule forceps with horizontal cystotomy	19	2	2	1
Vertical cystotomy, single or double, and blunt forceps	11	3	1	
T cystotomies	7	3		
	200	17	8	1

Secondary operations: 26 or 13%.

Analysis of these cases shows that if the capsular opening is a sufficiently large one (blunt or toothed forceps), as was obtained in 163 cases, secondary operation was necessary in 14. The addition of a peripheric cystotomy, while it facilitated the extraction of the cataract, increased the after cataract operation to 5 to 14 cases. The lateral vertical cystotomy, single, to facilitate grasping the capsule with the blunt forceps, and removing a large piece, or the two lateral vertical cystotomies with the attempt to remove the intervening capsule with the forceps, were not successful in reducing the frequency of the after cataract to less than 4 in 11 operations. Finally, the T shaped cystotomy gave the largest proportion of secondaries, 3 out of 7.

Analyzing the cause for the opaque membrane from another angle, tho I am fully aware of the difficulty of accuracy in this, we find the following:

After Cataract 26.

Retained cortex	8
Uniform thickened membrane, or one or more bands	15
Iridocyclitis	3

A final word about the most important complication of capsulotomy operations, namely, glaucoma. Collins particularly has shown that glaucoma develops from adhesion to or entanglement of the lens capsule in the extraction scar. This holds the anterior of the ciliary processes against the posterior surface of the cornea, blocking the angle of the anterior chamber. This is sometimes caused by inflammatory exudate, which, in contracting, produces the same effect. The amount of advance in the lens capsule depends on the position of incision; thus, in adhesion of lens capsule to limbal incision, glaucoma is rare. Adhesion of the hyaloid to the corneal scar may have the same result.

In this series of 200 capsulotomy cataract extractions, glaucoma occurred in 6. A table of these is as follows:

Glaucoma 6.

Capsular adhesions	3
Retained cortex, iridocyclitis	1
Malignant glaucoma	1
Retained cortex, capsular adhesion	1

Relieved by operation: 4. Eyes lost: 2.

To quote further from the above mentioned paper: "I know of no condition in cataract work which is more dangerous to the eye and more difficult to treat than postoperative glaucoma. Certain eyes are predisposed to capsular trouble, such as small hypermetropic eyes. An incorrectly performed section here exacts a terrific toll, for in those eyes the section must be scleral. The ideal result after the capsulotomy operation is that the capsular membrane should be deeply placed, with a clear, uniform space between it and the iris. Contact of the iris at the columns of the coloboma to the periphery of the cornea, due to adhesion of the lens capsule to the extraction scar, or adhesion of the capsule within the coloboma, are the usual causes. Glaucoma may, however, result from adhesion of the hyaloid membrane or of the vitreous to the corneal scar. One of the great advantages of the simple extraction and of the intracapsular extraction consists in the necessary avoidance of this condition.

In 1910, I reported on "Glaucoma from Adhesion of the Lens Capsule to the Cornea"[4] and I have been struck by the presence

of this condition in most of the cases which presented serious complications after cataract operations. If the adhesion is broad, and thickened by organized inflammatory exudation, the condition is very difficult to treat. A faulty corneal section is generally present. Iridectomy with division of the capsular adhesion gives relief for a time, but the condition generally recurs. Sclerectomy operations do not give the same good results that they give in noninflammatory glaucoma, and cyclodialysis has failed in my hands. In the early cases, and these cases must be operated on early, the adherent band must be divided or, better, resected, as in many cases there is a great tendency for the incision wound in the capsule to reunite. If this does not relieve the glaucoma, because the pathologic condition has led to organized closure of the filtration angle, a sclerectomy must be done.

The most important preventative is a properly placed corneal section. Next is correct technic of the capsule maneuver. Capsulotomy which avoids tags is essential. Careful replacement of possibly entangled capsule with the spatula, and freeing the corneal section of capsular tags at the close of the operation, usually prevents this unfortunate complication. A great step in advance would be a method of illumination by which the capsule could always be seen during operation."

References.

1. Arch. of Ophth., 1921, p. 115.
2. Ibid., 1915 and 1916.
3. Ibid., 1926.
4. Ibid., 1910.

DISCUSSION.

Dr. Harold Gifford, Omaha, Neb.: About thirty years ago, in an address on original research, I referred to the work of Herman Knapp, calling attention to the fact that the man who gives us the results of wide experience in a carefully analyzed series of cases, with the results on both sides, is doing as valuable original work as the man who glues his eyes to the end of a microscope for a large part of his life. I think we ought to congratulate ourselves on the fact that Arnold Knapp has followed so successfully in the footsteps of his illustrious father. The series of statistics which he has given us on this operation, which he devised and which he performs so successfully, is a very valuable contribution to our science.

I also want to note the value of this paper in its scope. A man who has a hobby like an intracapsular operation in one form or another usually puts on the loud pedal very forcibly. You will notice that Dr. Knapp says this is an operation which may be used with advantage in certain

selected cases, and gives full consideration to the common capsulotomy operation.

With regard to preliminary iridectomy, this is an operation which has caused much discussion. It seems like a good thing—the logical thing to suggest and carry out. Especially did this seem to be the case when, twenty or thirty years ago at the meeting of the British Society, a vote was taken as to how many of the members present would have a preliminary iridectomy if they had only one eye with a cataract in it, and every member voted in favor of this operation. If it is good for one eye, why is it not good for two? So for a year or two, I did a preliminary iridectomy in all my cataract cases. I gave it up, partly because at the end of this series I had an infection and lost an eye from a preliminary iridectomy; and second, because I found that I had an irresistible tendency to operate on these eyes too soon after the preliminary iridectomy, while the eye was slightly irritated. So I gave it up.

My experience further coincides with that of Dr. Knapp in the disadvantage of the operation in which the capsule is opened with a knife. Nearly everybody who does much cataract work sooner or later thinks he can cut the operation short by opening the capsule with a knife. It can be done, but I gave it up, partly because in a good many cases, I found it interfered with doing the section following. There is a little loss of aqueous, the iris may fall before the knife, and while you can cut out a piece of the iris with the knife without harm, you do not do the section as carefully as you should. Another thing, if you make the opening with a knife, you generally make a single vertical cut, which, as Dr. Knapp says, almost invariably leaves a cut thru the capsule with a thick band on both sides, and an insufficient opening.

Another method of preliminary capsulotomy was that of Drake-Brockman, who proposed doing the capsulotomy thru the limbus immediately before the cataract operation. He cut the capsule with a knife, and waited for the chamber to refill before going ahead with the operation. I tried this, too, but found that the chamber did not refill fast enough as a rule. It was a very nice thing when it worked well, because you can do your capsulotomy very much more accurately with the chamber full than with it empty.

If I believed, as Dr. Knapp does, in the extreme danger of capsule tags becoming pushed into the wound, I should do an intracapsular operation. In spite of the testimony of Treacher Collins, who has made an extensive study of this subject and found the capsule to be at fault in a large number of cases, I cannot believe that tags of the capsule are so dangerous as Dr. Knapp would lead us to believe. I believe, if we could section eyes in which we did an extracapsular operation successfully, we would find the capsule attached to the wound in a large proportion, just as Collins did in eyes that had to be removed. I do not look at the capsule at all after I once open it. I simply replace the angles of the iris and pay no attention to the capsule.

In regard to capsule forceps, nearly everyone has to try them sooner or later. Those with teeth did not give me a good opening, and when I took the cup shaped forceps that Dr. Knapp uses, and made a hole in the capsule in the center, I found I did not get a large enough opening.

I still had to do frequent discissions. Then I went down below the center and took hold of a large piece of the capsule, and that pulled the whole cataract out several times in succession. My horror of the danger of the loss of the vitreous was such, that instead of working it into a method, I gave up the forceps and went back to the cystotome. I fancy that a similar involuntary extraction of the lens was what led Dr. Knapp to take up his operation. Dr. Knapp, however, has developed it to a point which undoubtedly makes it the most successful of intracapsular operations.

Dr. Aaron Green, San Francisco, Cal.: There are a few points that I wish to stress in connection with Doctor Knapp's excellent paper. The first is in reference to safeguarding the patient. We tentatively choose the cases that are adapted for a certain type of operation when they are examined in the office, whether to be intracapsular or capsulotomy. The patient is given a sedative the night before operation, to secure a good night's rest. We safeguard our patients by paralyzing the orbicularis, and also by giving him an intraorbital injection of novocain, as described in a paper presented by us at the Minneapolis meeting. This is fortified by instilling cocain into the conjunctival sac. The novocain anesthetizes the intraocular structures and adnexa but it does not anesthetize the corneal or conjunctival epithelium.

Having prepared the patient in this manner, the next step is the incision. I do not think Dr. Knapp stressed that sufficiently. I think it is a great mistake to make an incision anything less than one-half of the corneal circumference. We have practiced that for fifteen years and have had no cause to regret it, but we have seen trouble from small incisions.

I also heartily approve of a complete iridectomy. I think it is poor surgery to try to extract a cataract without a complete iridectomy, no matter how widely the pupil is dilated. The pupil can never be as large as the lens, and to force the lens thru an opening smaller than itself is to cause trauma to the iris and brush off a certain amount of lens matter, which is apt to cause inflammation.

As to capsule forceps, I do not like the ordinary capsule forceps on the market. I agree with Dr. Gifford that there is some danger in taking out the lens in capsule by the use of a nontoothed forceps. On the other hand, I have found the multiple toothed forceps on the market to be dangerous. As Dr. Knapp suggests, it may require a great deal of pressure for the teeth to catch hold of the capsule. In our hands, no forceps has proved as satisfactory as Elliot's disc forceps that he uses for glaucoma. They are very sharp, and it is easy to get hold of the anterior capsule and pull it off like pulling off a piece of wall paper.

Many think that we perform the intracapsular operation exclusively. That is not correct. We perform the intracapsular operation only in about 65 to 75 per cent of cases. We have been able in the last ten years to make a definite decision as to the operation we will perform, but only after making the corneal section. The age of the patient, the prominence of the eyeball, tractibility and other signs may enable you to form a tentative opinion beforehand. But not until you have completed the section, can you say definitely which operation you will do. It all depends upon the appearance of the cornea. I have not seen this explained

anywhere in the literature, but we have used it for ten years as an absolute diagnostic point as to the operation of choice. It is very simple. After the section, you carefully examine the appearance of the cornea. If a horizontal crease appears on the cornea running from limbus to limbus, it is an absolute contraindication for an intracapsular operation. You will lose vitreous ninety-nine times out of one hundred, no matter what precautions you take. The reason for it is this: When the horizontal crease appears, it is due to increased intraocular pressure, which forces the lens against the cornea, causing a horizontal groove, with the lips of the corneal wound away from the sclera, and no matter what method you use, you will lose vitreous in an intracapsular operation. It is also a guide when doing a capsulotomy operation, as when you see this horizontal crease you would better complete the toilet rapidly and close the eye.

On the other hand, when the cornea drops down, or if there is a depression or dimpling in the center of the cornea, or if there is a vertical depression or crease instead of a horizontal one, you can safely perform an intracapsular operation. This is the final word so far as choice of operation is concerned. You need have no hestitancy in performing an intracapsular operation under these circumstances.

Another important point is the action of the lower lid in increasing or decreasing ocular pressure. The lower blade of any speculum is dangerous. The safest instrument for controlling the upper lid is Smith's lid hook, but it is difficult to make the incision and carry out all the steps of the operation with it. Anything that will elevate the upper lid but not retract the lower lid, provided it gives a large enough field of operation, is good.

About ten years ago, we removed the lower blade from our elevating speculum, and now control the lower lid only with the thumb. But even then, the lower lid should not be retracted after the incision has been made. Traction on the lower lid may cause the wound to gape, and may even raise the intraocular pressure sufficiently to cause expulsion of vitreous. Forward traction on the upper lid, however, causes negative pressure and minimizes the danger of expulsion of vitreous.

Dr. W. A. Fisher, Chicago: When Dr. Knapp reads a paper we expect, and usually receive, the last word, but there seems to be no last word upon this most important subject.

The majority of operators will agree with him, that it is no longer necessary to wait for maturity of a senile cataract before operating, and that the incision is one of the most important steps in cataract operation by whatever method selected.

If we could all remove a cataract with forceps as skillfully as Dr. Knapp, we would adopt that method as a routine, but those who do not operate in that manner should know that it requires great skill. Operators who have adopted a satisfactory technic do not feel like changing, and who can blame them? The question is, can they improve their technic without loss of many eyes. For many years I thought as Dr. Knapp, that a full iridectomy was imperative, but now I believe that a peripheral iridectomy is better.

Dr. Knapp states that peripheral iridectomy is not suitable for the

intracapsular operation, but if the eye is prepared as if a Barraquer operation is to be performed, peripheral iridectomy would be the proper procedure of operating by any of the intracapsular or by the capsulotomy method, because, as Dr. Knapp states, it materially reduces the percentage of iris prolapse.

It does not seem like Dr. Knapp to hear him state that he has not had any experience with the Barraquer method, and to follow the statement with a condemnation of the method on account of his belief that the pump is complicated. Such a statement coming from one so talented and so skillful, is one reason for others to condemn an operation that some operators believe the best surgical procedure so far proposed for the operation for senile cataract, and it is my belief that the Barraquer operation, with slight modifications, is within the grasp of any operator capable of successfully removing a cataract by any method.

Dr. Otis R. Wolfe, Marshalltown, Ia.: This splendid paper of Dr. Knapp's, along with the one he presented before the American Medical Association, should be a very convincing argument in favor of intracapsular extraction of cataract by some method. Luther Peter has also called attention to the capsule and lens debris as important factors in causing secondary glaucoma.

After five years experience with intracapsular extraction by phacoerisis, I am convinced of its superiority. The technic is more difficult but the results justify it. We favor a preliminary iridectomy a week or ten days preceding the extraction. With that exception, we use Green's modification of the Barraquer instruments and technic.

In selected cases, we endeavor to deliver the lens by the erisiphake alone. If it does not dislocate readily, we combine expression with it as described by Green. For this purpose we use a ball tipped forcep of our own design. (See Demonstration of Instruments, this volume.)

The superior visual results and freedom from secondary inflammation are very noticeable. In no instance have we had secondary glaucoma develop or loss of vision occur from any cause whatever, where the lens was delivered with the capsule intact.

We have, however, had it occur in cases where the capsule ruptured during the extraction. Our cases have all been private patients, and we have been able to keep in intimate touch with most all of them.

We hope to present our cases in satistical form at some future time, as Dr. Knapp has, and allow the results to speak for themselves. So far they have been extremely favorable.

Dr. Arnold Knapp, New York City, N. Y., (closing): I want to thank Dr. Gifford for taking part in the discussion. I think it was a great pity he was not allowed to talk more than the alloted time.

As to the capsule tags, which he does not fear, I would like to suggest, in all humility, that in his cases Dr. Gifford probably makes a peripheric section, in which the danger of glaucoma from capsular adhesion is much less.

I agree with Dr. Green, that the proper procedure in operating is to be able during the performance of the operation to decide on the proper procedure, or to switch from one type of operation to another, depending upon the indications present. I am interested to hear of his preference

for the forceps of Elliot, which I will be glad to try. I am not conversant with the crease on the cornea as a contraindication of intracapsular operation. I have noticed a line of opacity in the cornea after the intracapsular operation, which I regarded as being due to pressure of the upper lid.

In regard to Dr. Fisher, I am sorry I was too free in my condemnation of the Barraquer operation. My experience consisted in buying the instrument and taking it apart to try to see how it worked. I decided that it was a very complicated apparatus, and that if anything went wrong, it would be difficult to release the suction quickly, and I preferred to work with forceps controlled by one's fingers.

As for corneal suture, to be effective it must mean suture thru cornea and sclera. If you do this before the section, you will have difficulty in making your corneal section and conjunctival flap. Moreover, the removal of a corneal suture is always a difficult problem. In complicated cases I prefer to use the sliding conjunctival flap or apron which Dr. Harold Gifford suggested many years ago.

A FURTHER STUDY OF THE CULTIVATION OF LENS EPITHELIUM "IN VITRO."*

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In a former report[1] by one of us (Kirby), the object of our original investigation was stated to be a study of the nature of the cause of senile cataract and the possibility of its prevention or arrest by nonsurgical means. Various considerations showed that it was unwise to conduct such a research from the clinical side alone, as the same difficulties which have confronted other observers still remain unsolved from a scientific standpoint.

It was thought that much might be gained from a combination of clinical and fundamental laboratory studies. Attempts were to be made to study the living lens cells and to cultivate them outside the body. It was thought that living lens cells would form a suitable basis for research into the nature of the growth and nutrition and death of lens cells, after discounting the differences which would exist "in vivo" and "in vitro." The differential environment and the anatomic relations "in vivo" could not be reproduced "in vitro," but valid conditions for experiment might be created for lens cells as Carrel[2] had produced them for fibroblasts. Of necessity, we were forced to use the cells of lenses other than human for our research.

In one experiment[1] it was shown that the chick embryonic lens would live and differentiate "in vitro" in suitable media. Another experiment[1] showed that if the entire lens is dissected free from extraneous cells and cultivated in its capsule, no cells from within will grow outside the uninjured capsule. From a small number of these lenses cultivated "in toto," it was possible

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to get growths of living cells from the inside of the lens. These were proved to be from the anterior and equatorial epithelium. The cortex cells, representing devitalized cells, did not reproduce. Many cultivations of the lens "in toto" proved the efficiency of our method of dissection. Recognizing a lens "in toto" to be microscopically free from extraneous tissue after dissection, we could immediately with assurance make explants from its epithelium and later prove the cultures to be microscopically free from other cells. By a series of divisions of the original culture, a number of subcultures were obtained, so that a single strain was carried on thru seventeen generations over a period of six weeks.[1]

This laboratory work was discontinued for a period of one year in favor of a clinical investigation or study of standards for judging of the progress or arrest of senile cataract. We sought to learn of the natural history of untreated cataract in a large series of patients, so that we might have standards with which to judge cases which received treatment aimed at the prevention or arrest of the condition by nonsurgical means.

A report[3] of this study was published last year. This season, we have concerned ourselves with a further study of the behavior of lens cells when grown "in vitro." We will report further observations of their characteristics and also some controlled experiments which were conducted with them.

Percentage of Successful Original Explantations of Living Lens Epithelium.

Not all original explants of chick embryo lens epithelium prepared from fresh lenses lived and multiplied "in vitro." Depending on the condition of the cells and the suitability of the media, some cultures flourished, others just survived. A number of explants of epithelium showed a tendency to infold and join their edges and form miniature transparent crystalline lenses. These were often subdivided and active growths obtained in subcultures. Some original explants showed evidence of not having survived at all, while others died after a short period of survival.

In a period of eight months, 294 original explants of lens epithelium were made. Of these 174, or 59%, showed active growth. Not all of these withstood transfer to fresh media. Various reasons account for the failure of the 41% of the original explants to develope.

Percentage of Success With the Second and Third Generations of Lens Epithelium Cultures.

Lens epithelium growing "in vitro" forms a very delicate thin transparent membrane, which is difficult to remove cleanly from the plasma clot medium and transfer to fresh medium. The fragile cells tear readily and are injured frequently, so that they do not always survive. Of the 174 successful primary cultures, 112, or 38% of the 294 original explants, survived the second transfer. Of these, 56, or 19% of the original explants, survived the third transfer.

Difficulties Encountered in Cultivating Lens Epithelium.

It may be well to review here some of the difficulties encountered. Some of them pointed the way to success in cultivating the lens epithelium.

1. Embryos. Attempts were made to secure chick embryos of uniform size. The optimum size of eye was found to be about 3 mm., that of the lens slightly less than 1 mm. This size was usually secured by five days incubation at 37.5° Centigrade. Uniformity of size could not be assured. Many variable factors influenced the development of embryos in fertile eggs. Embryos of less or greater development usually showed protoplasmic adhesions between the lens and ectoderm or optic vesicle, which interfered with the clean removal of the lens.

2. Dissection of Embryos. The perfected technic described in a former paper gave a high percentage of success in freeing the embryonic lens of all extraneous tissue when the embryo used was at the proper stage of development. Lenses injured in removal usually showed nonviable cells.

3. Dissecting Fluid. Tyrode[4] solution was used. Because of some unexplained reason, Tyrode solution often changed a few days after its preparation from a pH of 7.8 and 7.9 to a pH of 8.1 or 8.2. Lenses dissected in the latter solution did not thrive afterwards.

Dehydration. Embryos were best dissected when freshly removed from the shell. If the embryo was allowed to become dehydrated by exposure to the air, deterioration of the eye tissues set in rapidly. The lens epithelium was found under these conditions to be granular, soft, fragile and without its normal elasticity.

Transfer of Cultures.

For the successful transfer of cultures of lens epithelium, it is necessary to remove the culture cleanly from the clot with a cataract knife, without transferring any of the media which has been digested and used by the cells for their nutrition. The body of the culture and much of the new growth must be transferred without injury to the delicate cells. This is difficult, because of the transparency of the thin membrane of tissue. It is not easy to see the edges. The frail cells are torn readily. The cultures are small. The average fragment explanted measures .25 sq. mm., while the average square area of the growth after 48 hours is 1.75 sq. mm. The handling of the delicate, minute cultures of lens epithelium renders necessary the development of a special technic.

Other Difficulties Common to Cultivation of Tissues.

The pitfalls and difficulties found with glassware, mica, water, plasma, extract and contaminations are common to the cultivations of any kind of tissue. We had a surprisingly small number of infections, less than ¼ of 1%, despite the fact that the media is excellent food for bacteria.

The Successful Cultivation in Unbroken Strain of Lens Epithelium Over a Period of Seven Months.

In a previous paper[1] it was reported that it was possible to carry on a strain of lens epithelium over a period of six weeks thru 17 generations. The cultures were thriving when the work was discontinued.

During the present period of research, 294 original explants have been made. A number of strains were developed from original explants and thrived thru a varying number of generations. One strain was started as an original explant, Culture No. 87-1, on November 28th, 1927. Of nine explants made that day, it was the only one which survived after four passages.

More often than not, the entire culture was transferred to new media each 48 hours, as this particular culture did not in its early life tend to grow large enough to be subdivided. After six weeks, it was possible to divide the culture more often. Then, due to unfortunate happenings and toxic influences of the plasmata used, the strain dwindled to one culture. Later, better results were had by subdivision. Early in June, there were

living six active cultures from the original explant. On June 28th, 1928, after 112 passages into new media, the last of the cultures were put into fixation to be preserved as stained specimens.

This particular explant did not provide as many subcultures for experiment as others did. Some other cultures would quickly grow to such a size that they could be readily divided and furnish two cultures.

This strain of lens epithelium demonstrated that lens epithelium "in vitro" can live for an indefinite period. It was grown in equal parts of adult chicken plasma and chick embryo (minus eyes) juice. The media was changed every 48 hours, except over the week-ends, when it was changed after a shorter period, or put on the shelf at room temperature for a period of 18-24 hours.

Cultures of Lens Epithelium Available for Controlled Experiments.

For experiments with tissues "in vitro," it is necessary to control the culture grown in experimental media with a similar or related culture grown in control media. Fibroblast cultures can be divided and two equal cultures obtained.[5] One can be used for experiment, the other for control.

After division of lens epithelium, the equal growth of the fragments could not be assured. It was found that better and more valid conditions for experiments with lens epithelium could be obtained by using two cultures which had appeared to have similar growth characters thru several generations. One of these was used as the control, the other as the experimental culture.

Despite the cultural difficulties, it was possible to have available on 69 experiment days (75% of the total number of days), over a period of seven months, sufficient cultures for a small number of controlled experiments. These were not always used, due to other important factors not being right at the time.

The average number of cultures available on each experiment day was 7. The greatest number available at any time was 15, the least being zero. In the latter case, there were always some cultures living, but thru some mishap they were in a sick or debilitated condition.

The Rate of Growth of Lens Epithelium "in Vitro."

For the purpose of comparison of the growth of lens epithelium cultivated in experimental media, it was desirable to have data on the growth of matched or related pieces of tissue when placed in normal or control media.

Fragments of tissue as minute as the cultures of lens epithelium, which when placed in cultures measure about .25 sq. mm., and after 48 hours growth measure about 1.75 sq. mm., could not accurately be weighed. They could not well be separated from the media. Nor could the cells which grow in transparent membrane formation be counted.

Recourse must be had to measurement of the square area of the fragment and later measurement of the growth surface area and comparison of the two. The method described by Ebeling[6] was used. Briefly, it is as follows: The culture fragment, before incubation, is placed in the projectoscope, magnified by 50 and its outline traced on paper. This procedure is repeated 48 hours later to measure the surface area of the growth. Difficulty was at first experienced in locating the delicate border of the new growth of the lens epithelium, but practice overcame this somewhat, so that the drawing could be made before condensation took place in the concavity of the culture glass.

The surface area was measured with the aid of the compensating polar planimeter. Results obtained in Vernier units were reduced to actual surface area and comparisons made. In the following tables are given the results obtained in measuring 96 cultures grown in control media. The surface area of the original fragment is compared with the surface area of the growth, and the relative increase expressed as the number of times the growth area is greater than the fragment area.

Rate of Growth of Lens Epithelium in Normal Control Media.

Experiment number	Sq. area of frag. in sq. mm.	Sq. area of growth in sq. mm.	Relative increase	Experiment number	Sq. area of growth in sq. mm.	Sq. area of growth in sq. mm.	Relative increase
208-1	.27	2.5	9.2	532-1	.15	1.5	10.
-3	.35	2.7	8.0	-3	.19	1.7	8.9
-5	.32	3.5	10.9	-5	.27	1.4	5.1
213-1	.32	2.2	6.8	548-1	.21	2.1	10.
-3	.35	3.6	10.2	-3	.26	1.6	6.1
-5	.32	1.5	4.7	-5	.24	.86	3.5
192-1	.38	2.5	6.5	575-1	.24	1.7	7.09
-3	.67	4.07	6.07	-3	.15	1.3	8.6
-5	.40	6.1	15.2	-5	.22	1.7	7.7
391-1	.23	2.8	12.2	568-1	.22	1.5	6.8
-3	.33	4.2	12.7	-3	.21	1.5	7.1
-5	.22	3.2	14.5	-5	.21	1.5	7.1
				581-1	.15	1.6	10.6
492-1	.24	2.1	8.7	-3	.13	1.9	14.6
-3	.22	1.4	6.3	-5	.24	1.4	5.9
-5	.17	1.5	8.2	590-1	.16	.94	5.8
497-1	.25	2.8	11.2	-3	.22	1.5	6.8
-3	.34	1.5	4.4	469-1	.16	1.7	10.6
-5	.31	1.6	5.4	-3	.22	1.5	6.8
413-1	.28	1.6	5.7	-5	.23	1.5	6.5
-3	.40	1.8	4.5	510-1	.16	1.3	8.1
-5	.32	1.4	4.3	-3	.23	2.2	9.5
519-1	.23	1.5	6.5	518-1	.27	1.3	4.7
-3	.20	1.9	9.5	-3	.23	1.5	6.5
-5	.22	1.5	6.8	-5	.24	1.6	6.6

Experiment number	Sq. area of frag. in sq. mm.	Sq. area of growth in sq. mm.	Relative increase	Experiment number	Sq. area of frag. in sq. mm.	Sq. area of frag. in sq. mm.	Relative increase
381-1	.41	3.5	8.5	527-1	.27	1.3	4.8
-3	.46	1.7	3.6	-3	.21	1.5	7.1
-5	.27	2.5	9.2	549-1	.24	1.4	5.8
385-1	.30	2.7	9.0	-3	.13	1.4	10.7
-3	.27	2.6	9.6	-5	.16	.87	5.4
-5	.25	3.1	12.4	538-1	.23	1.5	6.5
322-1	.24	2.0	8.3	-3	.15	1.7	11.3
-3	.26	1.7	6.15	-5	.24	1.8	7.5
-5	.23	1.5	6.5	556-1	.17	2.02	11.8
272-1	.23	1.2	5.2	-3	.24	3.1	12.9
-3	.23	1.9	8.2	-5	.18	2.1	11.6
-5	.29	1.5	5.1	567-1	.25	1.2	4.8
394-1	.23	2.04	8.8	-3	.24	2.2	9.1
-3	.16	1.5	9.3	-5	.24	2.4	10.
-5	.27	1.6	5.9	573-1	.15	1.5	10.
258-1	.26	2.6	10.0	-3	.24	1.5	6.2
-3	.15	1.1	7.3	-5	.15	1.6	10.6
-5	.32	1.5	5.0	581-1	.23	1.2	5.2
232-1	.35	1.4	4.0	-3	.13	1.7	13.
-3	.39	1.7	4.3	-5	.24	.74	3.08
-5	.67	2.5	3.7	589-1	.15	1.6	10.6
241-1	.36	1.7	4.7	-3	.22	1.6	7.2
-3	.39	1.3	3.3	-5	.16	1.2	7.5
-5	.37	1.7	4.5				

SUMMARY OF DATA ON RATE OF GROWTH.

SIZE OF FRAGMENTS EXPLANTED

Size of fragment—							
.15 sq. mm.	.20 sq. mm.	.30 sq. mm.	.40 sq. mm.	.50 sq. mm.	.60 sq. mm.	.70 sq. mm.	
Number explanted—							Total
21	38	25	10	0	1	1	96

Of the 96 explants made, 63 explants (65% of the total) had an average surface area of .25 sq. mm. They measured approximately .5x.5 mm.

SIZE OF GROWTH AREA AFTER 48 HOURS

Size of growth area—								
1 sq. mm.	1.5 sq. mm.	2.0 sq. mm.	2.5 sq. mm.	3.0 sq. mm.	3.5 sq. mm.	4.0 sq. mm.	5.0 sq. mm.	6.0 sq. mm.
Number of cultures—								
9	53	14	9	5	3	2	0	1

These figures represent the total area minus the area of the original fragments.

Of the 96 cultures, 67 explants (68% of the total) had an average surface area of 1.75 sq. mm. This was equal to a square having sides of about 1.325 mm., or to a circle having a radius of .75 mm. The cultures appeared to be larger than these figures would represent. The delicate finger like peninsulae extending out from the body of the culture left spaces between them that were not covered by the lens epithelial cells.

The relative increase of lens epithelium "in vitro" is obtained by dividing the surface area of the growth (total surface area after 48 hours minus the surface area of the original fragment) by the surface area of the original fragment. The average relative increase for lens epithelium in 96 cultures was 7.5 times.

SENSITIVITY OF LENS EPITHELIAL CELLS TO CHANGES IN THE CONSTITUENT INORGANIC SALTS IN NORMAL MEDIA.

It was important to know whether lens epithelium "in vitro" was sensitive to minor changes in the constitution of the media. Experiments were devised in which various inorganic salts normally present[4] were increased 25% over the normal. The effect of 25% increase in NaCl will be given in the Section on Hypertonic Media.

ACTION OF 25% INCREASE IN VARIOUS CONSTITUENT SALTS OF NORMAL MEDIA.

	Culture number	Relative increase in surface area	Ratio experiment to control
Control .02% KCl	208 -1-3-5	9.3	
Exper. .025% KCl	208 -2-4-6	6.74	.67
Control .020% $CaCl_2$	232 -1-3-5	4.0	
Exper. .025% $CaCl_2$	232 -2-4-6	2.90	.62
Control .020% $MgCl_2$	394 -1-3-5	8.0	
Exper. .025% $MgCl_2$	394 -2-4-6	6.7	.74
Control .005% NaH_2PO_4	272 -1-3-5	6.5	
Exper. .006% NaH_2PO_4	272 -2-4-6	6.8	1.04
Control .1% $NaHCO_3$	310 -1-3-5	4.9	
Exper. .125% $NaHCO_3$	310 -2-4-6	4.3	.87

The average of three experiments in each case is given.

In each series of experiments except one, the control cultures were better on the average, than the experimental cultures growing in the altered media. In one series, that of 25% increase in NaH_2PO_4 content, the experimental cultures were better than the control. This was not regarded as any specific stimulating action of this salt.

The results of these experiments would indicate that lens epithelium "in vitro" reacts in sensitive fashion to changes in the media.

THE EFFECT OF HYPERTONIC AND HYPOTONIC MEDIA UPON LENS EPITHELIUM CULTIVATED "IN VITRO."

A large series of experiments were conducted to determine the effect of variations of the osmotic tension of the media upon the growth of lens epithelium. This was thought to be important, for while the lens epithelium had previously been observed to grow best in normal medium, it had also been noted that normal medium was not isotonic for the lens cortex. A solution containing 1.2% NaCl had been found isotonic for the cortex. It was thought that lens epithelium grown in media having an osmotic pressure higher than normal might show different characteristics.

Microchemical methods were found feasible for qualitative detection of the various inorganic elements in the media, but we were at a loss for a quantitative method applicable to such minute amounts of material.

The best method of learning the exact osmotic tension of the media seemed to be the cryoscopic or the determination of the

freezing point. A micro-Beckmann apparatus suitable for use with as little as 2 c.c. of media was used. It was found that the presence of the albuminous material in the plasma and extract and the property of coagulation of the plasma interfered with exact determinations of the freezing point. Special experimental standards had to be determined. These worked fairly satisfactorily when the same plasma and extract were used for a large number of determinations, but we were dependent on different plasma and extract, which it was necessary to prepare frequently in small quantities. After four months trial of the cryoscopic method, it was abandoned. We returned to the direct measurement of the amount of salt or water added to the media.

Experiments.

The first experiments were designed to find the toxic points for hypertonic and hypotonic media.[6] It was proposed to add NaCl in measured quantities to the media until it became too concentrated for growth, and then in another series of experiments to add water until the media was too dilute for growth. Triple distilled water and conductivity water were used in the tests. To keep the same relative amounts of normal plasma and fresh embryo extract in the control and in the experimental media, Tyrode solution was added to the control media, to the same quantity as the salt solution in the experimental media The following proportions were used in all experiments.

Control Media	Experimental Media.
1 Part extract	1 Part extract
1 Part Tyrode solution	1 Part salt solution
3 Parts plasma	3 Parts plasma

Ebeling[6] had found that variations in the osmotic tension of the media from the normal at first tended to stimulate growth of the fibroblasts, and then after a few days always to retard it. It was not feasible for us to carry the experimental cultures thru a number of generations. We based our results on a comparison of the 48 hour growth of the control and experimental cultures, and the uniformity of the results, which showed the control cultures of the lens epithelium to be better than the experimental even when the media was varied only 25%, showed that the method was correct. If the cultures had been kept in the experimental media longer, they might have shown a more gradual curve toward the toxic point. The surface area figures were obtained as described in the section on "Rate of Growth."

ACTION OF HYPERTONIC MEDIA OF THE RATE OF GROWTH OF LENS EPITHELIUM "IN VITRO."

	Percent NaCl	Culture number	Surface area of fragment sq. mm.	Surface area of growth sq. mm.	Relative increase	Ratio of experiment to control	Average
			1% NaCl				
.8%	Control	192-1	.38	2.5	6.5		
1.0%	Exper.	-2	.62	3.8	6.1	.93	
.8%	Control	192-3	.67	4.07	6.07		
1.0%	Exper.	-4	.60	2.2	3.6	.59	
.8%	Control	192-5	.40	6.1	15.2		
1.0%	Exper.	-6	.30	4.7	15.6	1.02	.84
			1.2% NaCl				
.8%	Control	492-1	.24	2.1	8.7		
1.2%	Exper.	-2	.15	1.4	9.3	1.06	
.8%	Control	492-3	.22	1.4	6.3		
1.2%	Exper.	-4	.16	1.0	6.2	.98	
.8%	Control	492-5	.17	1.5	8.2		
1.2%	Exper.	-6	.13	1.5	11.5	1.4	1.14
			1.4% NaCl				
.8%	Control	497-1	.25	2.8	11.2		
1.4%	Exper.	-2	.28	1.3	4.6	41	
.8%	Control	497-3	.34	1.5	4.4		
1.4%	Exper.	-4	.35	2.0	5.7	1.29	
.8%	Control	497-5	.31	1.6	5.4		
1.4%	Exper.	-6	.48	1.4	3.0	.55	.75
			1.5% NaCl				
.8%	Control	413-1	.28	1.6	5.7		
1.5%	Exper.	-2	.27	1.5	5.5	.96	
.8%	Control	413-3	.40	1.8	4.5		
1.5%	Exper.	-4	.22	.54	2.4	.53	
.8%	Control	413-5	.32	1.4	4.3		
1.5%	Exper.	-6	.27	.77	2.8	.65	.71
			1.6% NaCl				
.8%	Control	519-1	.23	1.5	6.5		
1.6%	Exper.	-2	.22	1.5	6.8	1.04	
.8%	Control	519-3	.20	1.9	9.5		
1.6%	Exper.	-4	.21	1.9	9.0	.947	
.8%	Control	519-5	.22	1.5	6.8		
1.6%	Exper.	-6	.24	1.5	6.2	.91	.96
			1.70% NaCl				
.8%	Control	532-1	.15	1.5	10.0		
1.70%	Exper.	-2	.23	1.4	6.0	.60	
.8%	Control	532-3	.19	1.7	8.9		
1.70%	Exper.	-4	.17	.87	5.1	.57	
.8%	Control	532-5	.27	1.4	5.1		
1.70%	Exper.	-6		No growth		.0	.39

ACTION OF HYPERTONIC MEDIA ON THE RATE OF GROWTH OF LENS EPITHELIUM "IN VITRO."

		Culture number	Surface area of fragment sq. mm.	Surface area of growth sq. mm.	Relative increase	Ratio of experiment to control	Average
			1.8% NaCl				
.8%	Control	568-1	.22	1.5	6.8		
1.8%	Exper.	-2	.15	.34	2.2	.32	
.8%	Control	568-3	.21	1.5	7.1		
1.8%	Exper.	-4	.23	.98	4.2	.59	
.8%	Control	568-5	.21	1.5	7.1		
1.8%	Exper.	-6	.22	.73	3.3	.46	.45
			1.9% NaCl				
.8%	Control	581-1	.15	1.6	10.6		
1.9%	Exper.	-2	.15	.83	5.5	.51	Many dying cells
.8%	Control	581-3	.13	1.9	14.6		
1.9%	Exper.	-4	.14	.92	6.5	.44	do
.8%	Control	581-5	.24	1.4	5.9		
1.9%	Exper.	-6	.21	.92	4.3	.72	.42 do
			2% NaCl				
.8%	Control	469-1	.16	1.7	10.6	0	
2. %	Exper.	-2	.21		No growth		
.8%	Control	469-3	.22	1.5	6.8	0	
2. %	Exper.	-4	.19		No growth		
.8%	Control	469-5	.23	1.5	6.5	0	0
2. %	Exper.	-6	.19		No growth		

From the data given in the table, it is found that hypertonic media to the extent of 1%, 1.2%, 1.4% and 1.6% has an irregular effect on the cultures of lens epithelium. In each case, one of three cultures was stimulated to greater growth than the control, while two were depressed in their growth. In 1.7%, 1.8%, 1.9%, the effect was of fairly marked depression of the growth and the appearance in the cultures of signs of toxic effects. In some preliminary experiments, 1.75% and 1.9% proved lethal to one of the three cultures; 2% and any higher percentage of NaCl proved lethal to all the experimental cultures. The toxic point of hypteronic solutions for lens epithelium "in vitro" lies between 1.6% and 1.9% NaCl.

THE ACTION OF HYPOTONIC MEDIA ON LENS EPITHELIUM "IN VITRO."

To produce hypotonic media, triple distilled water was added to the media in gradually increasing percentage. Even in the experiments where 35% and 50% water were introduced, there was sufficient embryo extract present to stimulate the cells to

growth and to nourish them, and sufficient plasma present to form a good homogeneous clot. The factor, then, that produced the toxic effect was the hypotonicity or the lowering of the osmotic tension of the media.

ACTION OF HYPOTONIC MEDIA ON THE RATE OF GROWTH OF LENS EPITHELIUM "IN VITRO."

	Culture number	Surface area of fragment in sq. mm.	Surface area of growth in sq. mm.	Relative increase	Ratio of experiment to control	Average
5% Dilution with Water						
Normal	538-1	.23	1.5	6.5		
5% Dilution	-2	.25	1.5	6.0	.92	
Normal	538-3	.15	1.7	11.3		
5% Dilution	-4	.13	1.5	11.5	1.01	
Normal	538-5	.24	1.8	7.5		
5% Dilution	-6	.21	1.5	7.1	.94	.95
10% Dilution with Water						
Normal	549-1	.24	1.4	5.8		
10% Dilution	-2	.21	.73	3.4	.58	
Normal	549-3	.13	1.4	10.7		
10% Dilution	-4	.15	.97	6.4	.59	
Normal	549-5	.16	.87	5.4		
10% Dilution	-6	.21	.61	2.9	.53	.56
15% Dilution with Water						
Normal	556-1	.17	2.02	11.8		
15% Dilution	-2	.26	1.4	5.3	.44	
Normal	556-3	.24	3.1	12.9		
15% Dilution	-4	.30	2.0	6.6	.51	
Normal	556-5	.18	2.1	11.6		
15% Dilution	-6	.16	1.4	8.7	.75	.56
25% Dilution with Water						
Normal	573-1	.15	1.15	10.0		
25% Dilution	-2	.15	1.1	7.3	.73	Many dead cells
Normal	573-3	.24	1.5	6.2		
25% Dilution	-4	.21	.91	4.3	.69	Many dead cells
Normal	573-5	.15	1.6	10.6		
25% Dilution	-6	.13	.91	7.0	.66	Many dead cells, .69
35% Dilution with Water						
Normal	589-1	.15	1.6	10.6		
35% Dilution	-2	.16	No growth		0	
Normal	589-3	.22	1.6	7.2		
35% Dilution	-4	.23	No Growth		0	

ACTION OF HYPOTONIC MEDIA ON THE RATE OF GROWTH OF LENS EPITHELIUM "IN VITRO."

	Culture number	Surface area of fragment in sq. mm.	Surface area of growth in sq. mm.	Relative increase	Ratio of experiment to control	Average
Normal	589-5	.16	1.2	7.5		
35% Dilution	-6	.13	No growth		0	0

From these results, it is seen that 5% dilution with water has practically no stimulating or ill effect on the cells; 10% and 15% dilution have quite a marked depressant action on the growth; 25% had a greater effect than is evident from the ratio of the experimental to the control cultures, as there were many dead cells in the cultures. These cultures could not be restored by a return to normal media. Dilutions of 35% and 50% proved lethal to all experimental cultures.

ACTION OF MEDIA WITH HYDROGEN ION CONCENTRATION OF pH 8.2.

It had been noted frequently that Tyrode solution for some unknown reason tended to deteriorate, and its Hydrogen-ion concentration to change from a desirable pH of 7.8 or 7.9 to 8.2 or 8.4. Cultures placed in media made up with the latter did not thrive. Experiments were conducted to compare the effect of this slightly more alkalin media with the normal.

		Experiment number	Surface area of fragment	Surface area of growth	Relative increase	Ratio	Average
First Passage—May 26th, 1928—48 Hours Growth							
Control	pH 7.9	510-1	.16	1.3	8.1		
Exper.	pH 8.2	-2	.15	.87	5.8	.71	
Control	pH 7.9	510-3	.23	2.2	9.5		
Exper.	pH 8.2	-4	.27	1.6	6.6	.69	.70
Second Passage—May 28th, 1928—48 Hours Growth							
Control	pH 7.9	518-1	.27	1.3	4.7		
Exper.	pH 8.2	-2	.22	.86	3.9	.81	Many dead cells
Control	pH 7.9	518-3	.23	1.5	6.5		
Exper.	pH 8.2	-4	.19	.86	4.5	.69 .75	Many dead cells
Third Passage—May 30th, 1928—48 Hours Growth							
Control	pH 7.9	527-1	.27	1.3	4.8		
Exper.	pH 8.2	-2	.21	No growth		0	Dead
Control	pH 7.9	527-3	.21	1.5	7.1		
Exper.	pH 8.2	-4	.21	No growth		0	Dead

The effect of the slightly more alkalin media was to cause a fairly marked depression of the rate of growth in the first cultures. In the second cultures there were many dead and dying cells. The cultures were in poor condition and did not survive. All the cells were found dead in the third cultures.

The toxic agent in this media was evidently the altered hydrogen-ion concentration, the media being slightly more alkalin than normal, or it was the factor that brought about the change in the pH.

Toxic Influences on Lens Epithelium.

When toxic influences beset our cultures, affecting not only the lens epithelium but also the iris epithelium and the fibroblasts, it was often difficult to discover the exact factor at fault. The glassware was sometimes at fault, while at other times, it was the water used in making up the solutions; again the plasma or the embryo juice.

We tried to get along on as small quantities of material as was consistent with the experiments which we could conduct with our small staff. This small margin of safety was often a great handicap, as the toxic influence of some one factor would often debilitate or kill the cultures to such a degree, that it was several weeks before they could be revived and put into a healthy condition, ready for experiment again.

For this reason, it would be well to carry, if possible, a reserve supply of known nontoxic materials for use in any emergency. We were fortunate in reciprocating with another laboratory doing tissue culture work, so that at times we were able to avoid serious mishaps.

The toxic factor could often be discovered in a particular combination of media after a number of cultures had been debilitated or killed. At times, the effect was lethal, the cultures dying very quickly and not at any time showing any effort at migration or cell division. The central fragment became opaque. At other times, transparent cells and peninsulae of cells grew out from the central fragment more slowly and to lesser extent than in the normal healthy culture. This was apparently only a retarded rate of growth.

Other cultures showed that the individual cells were sick. The central fragment was more or less opaque, while the outgrowing cells were filled with refractile vacuoles and globules. The cells in the peninsulae retained their contact.

Other cultures showed a retarded toxic effect. Growing out in transparent peninsulae and membrane formation at first, the cells later separated one from the other and remained as isolated, rounded or spherical cells, unable to extend their protoplasmic processes.

Thus, lens epithelial cells under toxic influences "in vitro" may die "en masse" or as individual cells. Cells which have not lost their transparency may be revived. Probably some of the highly vacuolated cells may survive. Rounded, isolated lens epithelial cells are most probably dead. Further observations may lead to knowledge of the degree of debilitation of the cells from their microscopic appearance.

The Effect of Toxic Plasm on Lens Epithelium.

In a certain number of instances, it was possible to prove that the plasma contained the toxic factor.

Plymouth Rock chickens about one to one and one-half years old provided the best plasma when bled according to the Carrel technic. Certain debilitated birds gave plasma which proved toxic. Very young birds, or those which took the ether anesthetic badly, also gave toxic plasma. If the plasma gave off the odor of ether, it usually proved toxic. A certain chicken which had been inoculated with Rous chicken sarcoma virus quickly grew ill and its plasma proved to be toxic.

Lens Protein Experiment.

A Plymouth Rock hen, No. 002 (wt. 1.8 kilo.), was given in March, 1926, intraperitoneal injections of a centrifuged solution (Saline 4 to 1) of sheep lens protein. The dosage was increased daily, with the exception of two 48 hour intervals, from 2 to 20 c.c. Two days after the last injection, the serum showed a low potency in precipitin reactions. On April 7th, 1926, it received intravenously (wing vein) 5 c.c. of a solution of Beef lens protein (Saline 4 to 1). The next day 10 c.c. were given. In each injection some subcutaneous infiltration occurred. The following day, 18 c.c. were injected. On April 12th, 1926, blood withdrawn from the wing vein showed a positive precipitin reaction up to 1-5000 for Mulford's lens protein solution. Six days after the last injection, blood was taken from the carotid artery and plasma prepared in the usual manner for culture work.

This experiment was conducted to provide a serum which would give a marked tissue specific precipitin reaction and high

titre in complement fixation reaction. It was thought that such a serum would contain bodies which would react with substances in the cells of lens epithelium grown "in vitro" and kill them specifically, having no such ill effect on fibroblasts or iris epithelium. It was also thought that this reaction would demonstrate whether or not tissue specific lens protein was produced by lens epithelium grown "in vitro."

The media made up with the plasma from this chicken, No. 002, proved lethal not only to lens epithelium, but also to fibroblasts and iris epithelium. No specific effect on the lens epithelium was found. Some tremendous toxic factor had been produced. This experiment should be repeated several times, varying the conditions to make sure of the result.

The Resuscitation of Certain Sick and Debilitated Cultures of Lens Epithelium.

Many experiments which showed the toxic effects of various agents in the media were conducted against our desires in the matter. Striving for the production of healthy cultures with which to conduct certain projected experiments, we would often find the cells in a sick and debilitated condition on the day for the experiment. The central area would appear more or less opaque. The individual cells would become vacuolated, and those in the periphery would lose contact with the neighboring cells. By immediate transfer to normal media and by nursing thru a number of passages, the cultures were often saved. The dead cells would disappear, the cells in the peninsulae would again appear in opposition to each other. The vacuoles would no longer be seen and the culture would regain its healthy, transparent appearance.

Having learned from Dr. A. W. Fischer[7] that it was possible to keep cultures of other cells at room temperature for varying periods of time without much deterioration, we tried it with lens epithelium. After 24 hours incubation at 37.5° C, the cultures were occasionally placed on the shelf at room temperature for 24 to 48 hours. Then they were transferred to new media and reincubated. The value of this procedure is in the possibility of saving the strain when the other cultures become debilitated or die.

Summary and Conclusions.

1. In a further study of the characteristics of lens epithelium cultivated "in vitro," it has been possible to verify the experiments and observations previously reported.

2. Of 294 original explants of lens epithelium, 59% showed signs of active growth.

3. Of the 294 original explants, 38% survived the second transfer, and 19% the third transfer.

4. Lens epithelium "in vitro" forms a delicate tissue, which is difficult to handle and transfer.

5. Despite the difficulties, it was possible to carry a single strain of lens epithelium thru unbroken generations by 112 passages over a period of seven months.

6. The average explant measured 0.25 sq. mm., and the average growth 1.75 sq. mm. in surface area. This gave a relative increase of 7 times.

7. Lens epithelium "in vitro" is sensitive to changes in the inorganic salts in the media. It is best cultivated in media containing the normal percentages of inorganic salts. Hypertonic media containing from 1% to 1.6% NaCl stimulates some cultures and depresses others. From 1.6% to 1.9% it becomes progressively more toxic. Media containing 2% NaCl is lethal to the cultures. Hypotonic media in all except the very lowest dilutions has a marked depressant and toxic action on lens epithelium.

8. Media with an hydrogen-ion concentration of pH 8.2 was markedly toxic to cultures of lens epithelium.

9. In a certain number of instances, the adult chicken plasma was found to contain a factor which was found toxic not only to lens epithelium but to other cells.

10. It is possible to revive certain sick and debilitated cultures of lens epithelium by placing them in normal media for several passages.

11. Certain cultures of lens epithelium were kept in viable condition at room temperature for 24 to 48 hours after 24 hours at 37.5° C. The value of this procedure lies in the possibility of saving the strain when the other cultures become debilitated or die.

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DISCUSSION.

Dr. Walter B. Lancaster, Boston, Mass.: I happened to be on the committee at the time Dr. Kirby was appointed Research Fellow. Doubtless it is for that reason I am asked to open the discussion. The subject is not one with which I have had any personal experience. I did visit the laboratory and see the work that is being done by Dr. Kirby. Speaking in general terms, therefore, I think the most important point to bring out is that the Academy is here taking up a matter of pure research. Perhaps there are some who raise the question of what practical use is such research. It takes a little imagination to see practical results from these studies, but I should like to quote from an address by Doctor Robert A. Millikin in Science, September 18, 1928: "All knowledge that helps toward an understanding of the nature of the universe of which we are a part is useful, and we need very much more of it than we now have, or shall have for centuries to come, to enable us to direct our energies toward wise, effective living, instead of wasting them on beating tom-toms (certain methods of treating cataract) inventing perpetual motion machines, or chasing either physical or social rainbows." He then points out, that the recent exact measurement of the amount of lead in Black Hills uraninite, and of the exact atomic weight of that lead, is likely to have in the long run a much larger influence upon human conduct than the invention of the airplane or of the radio, important and preeminently useful though these be. I refer you to his article for his explanation of this statement. If you will read that paper you will see that he has forcible reasons for his conclusions:

It may well prove that studies of the properties of growing cells in vitro will lead to far more important conclusions than we can now understand. Such certainly was the case with discovery of Koch's method of studying bacteria by cultivation.

I think the Academy is to be congratulated on taking up important work of this sort and supporting it.

Dr. John M. Wheeler, New York City, N. Y.: I think that the Academy is to be congratulated on the work of this Research Fellow in Ophthalmology. Dr. Kirby has put himself in a forward position in this line of work. I think that the members of the Academy, or many of them, fully appreciate the importance of Dr. Kirby's being the first to grow the cells of the lens outside of the human body. He has made more of a study of the cultivation of lens fibers outside of the body than anyone else, and he proposes to go ahead with this study.

Dr. Kirby has a film which Dr. Carrell was kind enough to loan him, which he will show in closing.

A CONSIDERATION OF THE LATE OPERATIVE RESULTS IN GLAUCOMA.*

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PART I.

HISTORICAL.
THE NATURE OF GLAUCOMA SURGERY.
PUBLISHED RESULTS.

Glaucoma is a condition which falls within the domain of surgical ophthalmology. A more definite etiologic basis may be revealed in the future thru the experimental work of the physiologist and the biologic chemist, or thru the observations of the pathologist and the clinician, but until then, this opening statement is the vade mecum of glaucoma. We can compare our most wretched of modern aggregate operative results with those of 1842, wherein Sichel wrote, "Il n'existe point d'exemple avéré de guérison du glaucoma, etc:" a terrible prognosis, the triumph over which was reserved for Albrecht von Graefe (than whom there has been none greater), and his iridectomy intervention. In 1830, Mackenzie had struggled vainly with repeated paracenteses of the tunics of the eye to relieve the eyeball of its overfullness. In 1848, Desmarres attempted a pure anterior chamber paracentesis. In 1855, von Graefe presented his rebuttal of Sichel's pessimism when he said, "this overfullness was no mere complication but the CAUSE of all the leading symptoms in the disorder." Furthermore, he said something which Schweigger reiterated almost verbatim in 1891, "and the time honored rule is maintained, that as soon as glaucoma is definitely diagnosed, even tho in its earliest stages, iridectomy should be performed." True, the statement referred to acute glaucoma, for the treatment of the chronic types was then an

* By Invitation.

even greater problem than now, since the advent of the filtration cicatrix.

In the years 1873 to 1876, when Leber, Max Knies and Adolph Weber, all independently of each other, showed the relationship between the intraocular pressure and the aqueous angle of the anterior chamber, the necessity for operative intervention was fully recognized. Countless numbers of operations were presented in answer to the cry of the ophthalmologists.

Max Knies, in 1892, outlined the following dictum as fundimental for treatment (his nomenclature is a bit ambiguous):

1. Sclerotomy, posterior, for glaucoma of the congestive type.
2. Irisscleral puncture for cicatrical glaucoma.
3. Iridectomy and iridectomy with sclerotomy for glaucoma with occlusion of the pupil.
4. Iridosclerotomy for spontaneous glaucoma.

This last procedure was similar to that of Nicati's, called by him in 1894 scleriritomy. In this, a von Graefe knife was used for a subconjunctival puncture and counterpuncture combined with an iridotomy. Nicati called this iridien neurotomy—the purpose being to establish a filtrating subconjunctival cicatrix. In 1891, Snellen, speaking of glaucoma surgery, in general, said, "Should it be proved, as I think it will, that the essential part of the operation is the scleral wound, it still may be necessary in many cases to combine an iridectomy with it. In 1912, quoting Casey Wood, "Treacher Collins found, after a careful examination of eyes upon which iridectomy had been performed for glaucoma, that it is extremely rare for the initial section to pass thru the pectinate ligament, while Schlemm's canal invariably escapes." In acute glaucoma, where there is no iris-scleral adhesion, iridectomy may be truly peripheral. When adhesions have occurred, however, at this scleroiridian angle, the iridectomy cannot be peripheral, because of the anterior synechia of fibroblastic and round cell tissue deposition. Hence, Schlemm's canal is as tightly closed as before. Again quoting Wood, "In all likelihood, many cases of relief or cures following iridectomy are those due to the formation of cystoid scars or of minute fistulæ, rather than as a result of the removal of a portion of the iris periphery.

In the development of the trephining operation, we must not forget that de Wecker coined the words, "cicatrice à filtration" tho von Graefe expressed desire for scleral wound filtra-

tion. Stellwag, de Wecker, Mauthner, and others, in the years 1867 to 1880, recommended sclerotomies. Argyll Robertson first described his trephining operation in 1876, a scleral disc removal but posterior to the ciliary body—a procedure which Blanco and Frölich carried on. Legrange described his anterior filtrating sclerotomy with sclerectomy in 1907. Freeland Fergus and Elliot, in 1909, separately presented their anterior route scleral trephining, the former being a combination of trephining and cyclodialysis. Bjerrum, Herbert, Abadie, and Querenghi also presented filtration type sclerectomies. Holth, of Christiania, quoted by E. Temple Smith, in 1909, put forward the obiter dictum that, "Anterior subconjunctival sclerectomy is destined to be the basis of all future surgical treatment of chronic glaucoma." Heine of Breslau described his cyclodialysis operation in 1905, giving credit to Axenfeld and to E. Fuchs for the observations and the theories upon which he based the operation. Colonel H. Herbert (then Major), in 1903, presented a series of 130 cases operated with the production of a subconjunctival iris incarceration (iridotasis). In addition to these men cited, speaking only from the standpoint of early historical note and of basic technic development, the names of Vacher, de Lapersonne, Bettremieux, Brooksbank, and many others, must be included.

In 1913, Casey Wood said, "The names of glaucoma operations is legion." Of those he then mentioned, many are now only of historic interest, stepping stones to our present day methods. There are the basal and the peripheral iridectomies, and with the iris and sclera together, we have, or have had, iridosclerotomy, iridenkleisis, transfixation of the iris, dialysis of the iris, peripheral iritomy, scleroiridectomy, the wedge isolation operation, and other iris inclusion operations. The attack thru the sclera includes the anterior and posterior sclerotomies, anteroposterior sclerotomy, equatorial sclerotomy, T shaped sclerotomy, the Lagrange sclerectomy, punch forceps sclerectomy, Holth's cystoid cicatrix, angle incision (de Vincentiis' incisio iridian), and the Fergus and Elliot trephining operations. The sclera, iris, ciliary body and choroid, when considered together, have added division of the ciliary muscle, cyclodialysis, iris-ciliary-body-scleral puncture, ciliarotomy, hyposcleral cyclotomy, and scleral choriotomy. In addition the removal of both (or either) the ciliary ganglion and the cervical ganglion have been recommended.

But Priestly Smith, Snellen, Elliot, and Wood have covered the history of glaucoma surgery prior to and contemporaneous with their writings in a manner impossible to duplicate, nor is it relevant here. The reason for this preamble of history and technic is only to present the ancient precedent for the conviction held that glaucoma therapy is essentially surgical.

The value of miosis and miotics cannot be belittled in any way. Their timely use has prevented much irrecoverable blindness. They must be considered merely as a crutch, however essential it may be. Laquer of Strasburg, in 1876, decided that miotics and iridectomy are integral for threefold reasons:

"a. Without harm, and often with advantage, the operation, iridectomy, could be postponed to a more convenient time.

"b. Facilitate the operation.

"c. The effect of the operation is sustained by the miotic action."

It is impossible to deny the fact that many cases of glaucoma are carried on for many years, even for life, by miotics alone. This fact is also true, many more have lost vision by denying them surgical intervention when this could have been given. A life time of miotics is a Damoclean existance with blindness ever impending.

In 1913, de Schweinitz discussed in a classical paper the nonsurgical measures for the reduction of increased intraocular tension. Then, he said, "True as this is," speaking of surgical measures, "we are not yet in a position to discard nonsurgical procedures, (1), because operation is not always possible, (2) because operation is not always permitted, and (3) because in certain circumstances operation is not advisable." The measures recommended by de Schweinitz were, "(a) miotics, (b) reduction of tension by massage, electricity and diathermy, (c) indirect reduction by the lowering of general vascular tension, (d) reduction by the stimulation of osmosis, of lymphagog activity, of absorption of edema, and capillary contractility, and by decreasing the affinity of ocular colloids for water." These are mentioned here in this surgical paper as relevant, in that the combat against glaucoma demands every weapon at our command, for strange and unusual developments continue to make each case an individual problem.

The nature of surgical intervention may be shown fairly well by the following list of cases, abstracted from the clinical

records of the University of Pennsylvania Graduate Hospital. The cases extend over a period of 1923 to January 1st, 1928, they being drawn, mainly, from the clinics and the private patients of four different ophthalmologists. The total number is 291. No attempt has been made in this series to illustrate the end results. The series is given merely to show the type of operation which has been done and the diagnosis with the numbers of the different varieties of glaucoma operated upon. It was impossible to give a more accurate subdivision of the classes from the records alone. This was especially noticeable in attempting to differentiate the enucleations of absolute glaucoma from the enucleations which became necessary for failures in recovery following earlier surgery. True, these latter cases may have been in an absolute glaucomatous state also, but complicated by reason of unsuccessful surgery. Hence, almost all of the enucleations seemed to gravitate, unwittingly, into this subdivision.

Operations which were performed in the above mentioned group of cases.

Operations	Number of Cases
Absolute Glaucoma.	
Enucleations	34
Trephining	1
Iridectomy	1
Herbert's operation	1
With staphylomata	
Posterior sclerotomy	2
Iridectomy	3
Bilateral	
Bilateral enucleation	1
Chronic Glaucoma.	
Iridectomy	71
Trephining	65
Iridectomy with posterior sclerotomy	6
Cyclodialysis	5
Sclerectomy	5
Anterior sclerotomy alone	13
Herbert's operation	17
Anterior sclerotomy, later enucleation	2
Inflammatory	
Scleral trephining	2
With intraocular neoplasm	
Enucleation	3
With retinal detachment	
Multiple scleral puncture	1
Acute Glaucoma	
Iridectomy	16
Posterior sclerotomy with later iridectomy	3
Posterior sclerotomy with later trephining	2
Hemorrhagic Glaucoma	
Iridectomy	1
From thrombosis of central artery of retina	
Enucleation	1

Glaucoma with Senile Cataract. (Difficult to say if glaucoma was primary or secondary.)
- Iridectomy 4
- Iridectomy with later lens extraction..... 1
- Iridectomy following a posterior sclerotomy..... 2
- Trephining with later lens extraction..... 3

Secondary Glaucoma, secondary to uveitis or to trauma.
- Scleral trephining..... 3
- Iridectomy 2
- Cyclodialysis 2
- Lens extraction..... 2
- Posterior sclerotomy..... 2

Glaucoma, postoperative, after cataract operation.
- Cyclodialysis 2
- Iris inclusion 2
- Trephining 1

Buphthalmos.
- Iris inclusion 2

Excluding from these cases above, 66, for obvious reasons, as enucleations, etc., a total of 225 cases are to be grouped as follows:

Iridectomies	110	or 48.8%
Trephining	79	or 35.5%
Herbert's operation	17	or 7.5%
Cyclodialysis	9	or 4. %
Sclerectomy	5	or 2.6%
Iris inclusions	4	or 1.6%

From this above recapitulation, several very important points can be decided. Peter states that 6% of all the new cases in his private practice are cases of glaucoma. Wright, quoted by Peter, states that 7% of 3,705 new admissions to the Madras Ophthalmic Hospital in 1926 were cases of glaucoma. Estimate these 291 cases above as a 4% portion of clinic and private practice. Wright gives 2.6% as the percentage of glaucoma diagnosed in his out-patient clinic. It is a fact that glaucoma is diagnosed less frequently in clinic patients than in private practice, regardless of the reasons which may and do underlie this (and eliminating 31 cases herein which were reoperations): the total cases represented by this glaucoma index or incidence (260) is 6,500 new patients. If these rather approximate figures may be given credence, then also, the statement may be made "that surgery seems to be a treatment used in a very large percentage of these cases."

Second, glancing at the above figures, one sees too many enucleations for this condition—42. Granted that a certain percentage of enucleations are inevitable thru the fault of the patient, there will thus be a larger incidence in clinic practice. No private practice ever yields this grist of failures. Fifteen per cent of enucleations, in all cases, is too high, every contingency possible being considered. This is a statement of fact and not a criticism. It indicates only too well the necessity for out-

patient or clinic patient education. It shows a necessity for earlier diagnosis, or maybe for even more convincing arguments for surgery early in the course of a patient's attendance at a clinic. It also considers the prophylactic trephining upon the apparently normal eye in a patient with unilateral glaucoma. Blaauw and others have shown that the apparently normal eye always has a diminished light sense. How this educational program is to be achieved, if the above are applicable, is unknown to the author. The occurrence of blindness from glaucoma, these days, without any search for relief on the part of the patient is as deplorable as it is remedial.

Third, the recapitulation shows to the author a demand for a more accurate utilization of glaucoma nomenclature, especially in recording diagnoses on permanent records. It may be that many clinics are at fault in this. Acute glaucoma is too often unmodified. The case under question may be an acute congestive type in its primary attack, or recurrent, in an interval period, and discovered by tonometric readings and the fundus and field studies of a careful clinical ophthalmologic examination, and then called acute by reason of its apparent anamnesis. This same case, also, might be the first discovery of the long standing prodromal stage of chronic noninflammatory glaucoma. A similar error can be made in confusing chronic inflammatory glaucoma with acute congestive glaucoma in perhaps a recurrent phase, the former being correct in the instance. Correct diagnosis we know demands accurate examination and then, later, as careful a survey of all the data available, considering the central visual acuity still intact, the amount and variety of paracentral and peripheral field loss, the condition and the appearance of the nervehead, the nutritional state of the iris, the gross appearance of the eyeball, the average mean and extreme of repeated tonometric readings, as well as the general history of the case. But sufficient has been said on this point. To continue would not add emphasis.

Fourth, one of the questionnaires upon which many of the statistics in this paper are based was received from the chief of one of these clinics It will, therefore, apply to the results of a portion of the cases. These same results are embodied, later, in the grand totals, but they are peculiarly relevant here. These show but two reoperations after trephining, with at least 80% of the cases freed from later miotics (the real criterion of success), and with but one eye lost by infection and one other

enucleated. With the iris inclusion operation, a much smaller number of cases, there was one enucleation, and all were continued with miotics after the operation. The cases of iridectomy had a goodly number of reoperations subsequently necessary, and miotics were continued in all cases. Considering these three alone, there is little wonder that this same ophthalmologist saw fit, recently, to present the following relative to trephining before a western ophthalmologic society. "The advantages are many. First: in my experience, the highest percentage of good results are obtained by this operation. Second: there is no danger, barring accidents, of loss of the visual field or of central visual acuity. Third: the small pupil gives the patient more comfortable vision than a mutilating broad iridectomy. Fourth: the operator has a feeling of security that central vision and the visual field will be preserved indefinitely, even tho the patient may grow careless and cease to report. Fifth: it is applicable to a larger number of patients than any other form of operation."

While this paper is primarily a compilation, the author is giving here some personal experiences. By reason of association in the above mentioned clinic, part of these experiences will overlap those already mentioned immediately above and still earlier in the compilation. Considering trephining postoperative results, three enucleations have been done, one, an absolute glaucoma (the opposite eye held in normal tension with 6/6 vision following trephining), enucleated because of hyperexotropia with amaurosis. The second case was also in absolute glaucoma following a trephining operation with postoperative normal tension and a good filtering scar, but wherein the eye continued painful and tender for several months by reason of a low grade iridocyclitis. The third was a postcataract operation glaucoma, neglected for a long time, with total blindness and with early staphylomatous development. The cataract operation had been done in a foreign city clinic. Two cases have had a trephining operation repeated. A third remained normal following a cyclodialysis operation. Posterior sclerotomy has been used several times, successfully, to precede a trephining operation in acute congestive glaucoma. In one such instance of acute congestive glaucoma from thrombosis of a retinal blood vessel, the subsequent central vision was 6/7.5, with a relatively small field defect remaining. A wide basal iridectomy, when done early, has been sufficient usually to hold tension in the secondary glaucoma of immature cataract. Those cases of cataract complicated

by primary glaucoma, chronic type, have been best treated by an initial corneoscleral trephining operation, with a subsequent lens extraction, the cataract section being wholly corneal and anterior to the filtering bleb. Anterior sclerotomy (anterior chamber paracentesis) has proven of great value in certain instances of acute congestive glaucoma with uveitis, in connection with the use of miotics and prior to a later iridectomy. Iridectomy has often been the operation of compulsion for acute congestive glaucoma with uveitis. Paracentesis is preferable, but iridectomy may be necessary. In one case, several days after the iridectomy, anterior chamber paracentesis seemed to be the turning point toward recovery. In this case, the right eye being sightless from a neglected glaucoma, a most severe rhinitis precipitated the glaucomatous attack, this in turn being complicated by severe plastic iridocyclitis.

Secondary glaucoma follows a delayed healing after cataract extraction with a most distressing regularity. Peter first focused the author's attention on this fact by his recommendation for an immediate reoperation in these cases. An iris pillar prolapse, a delayed anterior chamber closure or reformation, a gaping corneoscleral wound or an incomplete anterior chamber toilet occasionally follow cataract operations. The most common cause is a patient's misbehavior, with precipitant lens expulsion and vitreous loss. In such cases, all operators strive to close the lids and to stop all further manipulations as soon as is possible. It is by far the lesser of the two evils. Also, early in the postoperative convalescence, anterior chamber collapse may occur from stooping, coughing, stretching, even from lesser activities. These as well fall into this catagory. None of these cases had vitreous herniation into the anterior chamber. The immediate reoperation recommended and necessary is an iridectomy of the prolapse, completion of the toilet of the anterior chamber and a final conjunctival flap closure. This should be done, if at all possible, considering those of the first class, 6 to 8 days after the cataract operation; in those of the second mention, a reoperation immediately after the development of this complication. In spite of this, a secondary glaucoma develops early in the convalescence of a small number of cases. It develops invariably in those not reoperated. Iris inclusion operations have been successful in about 50% of these complications. Cyclodialysis was successful in the two instances used. Corneoscleral trephining, at about 5 o'clock on the limbus, has been universally successful in all instances where used.

We now have a series of these cases under observation, awaiting a sufficient interval following the surgery for the anterior chamber collapse and the glaucoma before they may be reported in detail—the basic etiology, the complication which occurred, the reoperation and the ultimate results. Up to the time of this writing, the immediate reoperaion has proved justifiable.

Before further consideration is made of the statistics gleaned from the questionnaires sent out to various ophthalmologists, it might be well to mention authoritative opinions and results relative to the five major groups of operations, i. e., iridectomy, Lagrange sclerectomy, iris inclusion (iridotasis), trephining, and cyclodialysis.

A survey of the iridectomies, taken from Weeks' Diseases of the Eye follows:

Sulzer. Acute glaucoma	149 cases	73.5%	Improved
Zentmayer and Posey. Simple glaucoma		60. %	Improved
	Vision unchanged	20. %	
Wygodski. Inflammatory glaucoma	37 cases	76. %	Improved
Chronic glaucoma	147 cases	10. %	Improved
	Uunchanged	40. %	
	Failure	50. %	
Simple glaucoma	104 cases	.9%	Improved
	Deterioration	52.0%	
	Unchanged	10.5%	
	Amaurosis	36.5%	

Hahnloser and Sidler. 172 eyes observed not less than 10 years after operation:

Acute inflammatory	31 cases	64. %	Improved
	Relatively good	13. %	
	Blind	23. %	
Chronic glaucoma	37 cases	29.9%	Improved
	Relative good	27. %	
	Blind	43. %	
Simple glaucoma	76 cases	42. %	Improved
	Relative good	28.9%	
	Blind	28.9%	

Meller's survey of 389 Lagrange sclerectomies done by himself showed the following end results:

Acute inflammatory glaucoma	12.0% of cases
Chronic inflammatory glaucoma	61.5% of cases
Simple glaucoma	9.0% of cases
Recurrences	11.3% of all cases
Good results	70.0%
Poor results	10.0%
Lens involvement	4.0%
Iridocyclitis	2.3%
Enucleations	3.4%
Late infections	1.3%
Vitreous loss	6.2%

Of the recurrences, Simple glaucoma 14.3%; Chronic glaucoma 6.0%

It is of interest to note here that Lagrange reported, in 1927, as the contraindications to his operation acute glaucoma and

subacute glaucoma with a tension at the time of operation above 40 mm. Hg. (Schiötz).

Wilder, in 1923, reported a series of 48 iris inclusion operations, in only one of which disaster occurred. The operator, at the 1927 meeting of the Ophthalmological Meeting of the United Kingdom, in spite of the almost universal opinion against this operation, that it was unsurgical and utilized the vulnerable iris, stated, "From having practiced iridotasis as often as trephining (over 100 times), that one was as safe as the other, and of all the operations for glaucoma which he had done, the one which appeared to cause the least traumatism to the eye was the simple procedure of iridotasis. The eye recovered promptly from that operation, and with the least possible reaction and least damage to the parts. His series of 48 cases, presented before the ophthalmologic section of the American Medical Association, listed 10 cases of acute congestive glaucoma and 38 cases of chronic noncongestive glaucoma. Of these cases, 44 were followed by tension reduction, a 90% success.

In the congestive glaucoma group, in one blind eye, with a tension of 100 mm. Hg. before the operation, the tension was reduced to 45 mm. Later, an iridectomy brought the tension to the normal. In this congestive group, one case was normal after 7 years, one after 3 years, one after 2 and ½ years, three after 10 to 14 months, and 3 after 3 to 4 months. In the chronic, noncongestive group of 38 cases, one, an absolute glaucoma, had a tension of 40 after 6 months interval, a second was reduced to normal for 3 months and then rose to 40 mm. Hg. After an iridectomy, this dropped to the normal and remained there. A third remained at normal tension, but following a massive choroidal detachment, vision which had been 20/30 before the operation remained at 20/50. The fourth case was that of a streptococcus panophthalmitis with subsequent evisceration. Of these 38 cases, the remaining were as follows: 17 with normal tension after 3 years, 3 after 2 years or more, 3 after one year or over, and 12 have remained normal for less than one year. In 1923, as in 1927, Wilder stated that proper cases must be selected for the iris inclusion operation. He believes the best cases for this operation are those of noncongestive glaucoma, wherein trephining or sclerectomy might also be done. Those with high blood pressure and with indications of atheromatous blood vessels, wherein one might fear hemorrhage from the fragile iris vessels, and cases of unsuccessful iridectomies, tre-

phinings and sclerectomies, were especially applicable to the operation. The special contraindications are those cases with developing cataract, secondary glaucoma with synechiae, and acute congestive glaucoma. In this group, iridectomy still seems most satisfactory.

While our experiences have not been as happy with iridotasis as Wilder's, they do seem to improve late after the operation. We also agree without any doubt, the operation offers the least traumatism to the eye of any type of glaucoma operation, save that of cyclodialysis.

In view of the similarity that Holth's iridoencleisis operation has to iris inclusion, a series of these are also included. Holth, himself, reported two groups respectively of 45 cases and of 87 cases. The first group showed 85% results with good drainage and normal tension. The second group had 86% results with good drainage and with normal tension. Harold G. A. Gjessing of Drammen reported, in 1925, Acta Ophthalmologica, a series of 89 cases performed according to Holth's technic, extending over a period from 1911 to December 1924. The minimum time which had elapsed at the time of his follow-up check was 8 months, the maximum was 116 months. Of the 89 cases, 79 were thus reexamined. His findings in these 79 cases follow:

Preserved or increased vision.....	68 or 86%
Preserved or increased field of vision.....	62 or 78%
Normal tension	65 or 82%

His conclusions relative to the operation were: (a) the maximum result will be established within 6 months after the operation, (b) it gives a relatively certain and lasting result without exposing the eye to any considerable risk because—

1. When the operation is regularly performed no thin bleb is formed;
2. The corpus ciliare is never exposed to such an extent, as frequently occurs with the Elliot;
3. Both these factors tend to prevent late infection;
4. Hypotension is not seen,

and (c), the cosmetic result is good. The pupil is but slightly dislocated and blinding of the eyes is avoided.

Wilmer's series of trephining results presented in 1927 can well be the basis for the rapid consideration of this operation. His Tenon's capsule transplant refinement of the operation need

not be discussed here except for this bare mention. He reported 107 cases in two groups as follows:

A Period of observation. This was from 15 years to 3 years.
B Primary glaucoma.
C Secondary glaucoma.
D Complete iridectomy.
E Partial iridectomy.
F Postoperative iritis.
G Average tension before operation.
H Average tension after operation.
I Success.
J Failures.

Table 1.

A	B	C	D	E	F	G	H	I	J
	50	22	70.83%	29.16%	26.38%	47.3 mm.	14.9 mm.	84.7%	15.2%

Table 2

A	B	C	D	E	F	G	H	I	J
	28	7	91.4%	8.6%	8.57%	39.8 mm.	12.6 mm.	91.4%	Partial 5.7% Complete 2.8%

In Table 2 the period of observation ranged from 2 years to 4 months.

A total of these two above tables, average shows:

Success ..... 88.0 %
Partial failure 1.88%
Complete failure 10.14%
Late infection ..... .93% (Eye saved)

In the light of these results, it is of interest to note Elliot's answer on the questionnaire received from him. "I find trephining so satisfactory that I do no other operation in any form of case." He excludes in this the consideration of secondary or postuveitis glaucoma. Such a statement from this authority is of the highest importance.

The consideration of cyclodialysis is based upon a series of 51 cases (60 operations) presented by Schmidt of the Erlanger Universitäts-Augenklinik (Vorstand Prof. Dr. Fleischer), in 1927. This series of cases included the following—a group of severe cases showing rigid demands on the operation used:

Noninflammatory glaucoma ..... 29 cases
Inflammatory glaucoma ..... 3 cases
Glaucoma with high myopia..... 3 cases
Secondary glaucoma ..... 2 cases
Postcataract operation with incarceration of iris..... 1 case
Glaucoma with parenchymatous keratitis..... 2 cases
Glaucoma with old parenchymatous keratitis..... 2 cases
Glaucoma, old, free from hypertension for one year up to two days before operation..... 1 case
Glaucoma following previous operations..... 9 cases
After sclerotomy ..... 1 case
After trephining ..... 3 cases
After iridectomy ..... 5 cases

The results of the reoperation cases were as follows:

After sclerotomy .....Good result
After iridectomy

Simple glaucoma ..... 2 cases Inflammatory glaucoma.. 2 cases Secondary glaucoma .... 1 case	.....	1 case fair results, 4 cases good results, in one case a cyclodialysis repeated.

After trephining
One case trephined three times.....Good results
Two cases trephined once.....Good results

After the operation, the immediate fall in tension was:

Very marked in..... 38 instances
Small in ..... 18 instances
No change in..... 4 instances

The tension was again elevated as shown in the following:

After 2 to 3 weeks	1 case	1.8%
After 1 to 6 months	8 cases	15. %
After 6 to 12 months	1 case	1.8%
After 1 to 2 years	1 case	1.8%
After 2 years	2 cases	3.6%

These cases were followed up for the period of 1921 to 1926. Of those definitely checked:

Central visual acuity and fields of vision remained normal:
In 22 cases with normal tension.
In 8 cases with an increase in tension.
Central visual acuity and fields of vision decreased:
In 4 cases with normal tension.
In 7 cases with an increase of tension.

There was a definite success in all cases of from 45% to 50% at least. The table of recapitulation follows:

A Year.
B Successful.
C Failure.
D Remaining.

A	B	C	D
Glaucoma simplex			
1921.....	6	2	1
1922.....	3	2	—
1923.....	—	2	—
1924.....	6	—	—
1925.....	10	2	2
1926.....	7	1	—
Inflammatory glaucoma			
1921.....	—	1	—
1923.....	1	—	—
1926.....	1	—	—
Secondary glaucoma			
1922.....	1	—	—
1924.....	1	—	—

This small series was used rather than a massive series as that of Salus, with his group of 456 consecutive cyclodialyses. Hence Salus may have included cases which would have been,

by a large number of operators, treated with some other technic. This applies especially to the inclusion of inflammatory and juvenile glaucoma.

The accidents which Schmidt lists in his series were:

Partial iridodialysis	3 instances
Vitreous prolapse, slight	3 instances
Severe vitreous hemorrhage	1 instance
Moderate anterior chamber hemorrhage	Occasional
Partial denudation of Descemet's membrane	Occasional

The operation preceded to a close uneventfully in 53 instances. The slight anterior chamber hemorrhage, in those cases wherein it occurred, disappeared in 10 days. The one case with severe vitreous hemorrhage had an unsuccessful result. Those cases with partial iridodialysis, it seemed, were not harmed by this complication. This also applied to the tiny rolls of Descemet's membrane, which, when lifted, fell to the bottom of the anterior chamber.

Schmidt considers the operation a valuable one in the armamentarium of glaucoma therapy because of: (a) its ease of technic and absence, relative, of complications, (b) it can be repeated 3 or 4 times, (c) it does not render impossible other operations, as an iridectomy or trephine or even a lens extraction, and (d) it is not followed by a cosmetic defect.

PART II.

Specific Compilations from Received Questionnaires. Conclusions Logical with These Concrete Tables.

In this next section, we will consider, in table form, the end results of glaucoma surgery as determined from the copies received completed of an extensive questionnaire sent to various ophthalmologists in the States and abroad. In developing these tables, an attempt has been made to strike an average, in the results, save where the answers were so dissimilar that the differences in opinion had to be reported for the proper presentation of a qualification. For example, one physician reported the nonuse of miotics after trephining, while another reported an incidence as high as 100%. In this one point, alone, it might seem as if some cases were continued unnecessarily with miotics while others needed them. Each table will explain itself without an extensive preamble. Footnotes, unfortunately, will be necessary in a majority of these tables.

The first to be considered is a comparison of the 5 major surgical groups from the standpoint of reoperations, enucleations and the continued use of miotics following operation. Personal liking for trephining is the only reason for placing this first in the table. It looks as if the procedure has merited this place.

A word here relative to recent work in the raison d'etre of cyclodialysis is not amiss. Stoughtenborough recently, in the American Journal of Ophthalmology, presented a study of this operation based upon his observations in normal rabbit eyes. The older theory was, as everyone knows, that it opened the suprachoroidal space into the anterior chamber in a permanent intraocular fistula. Heine based his operation upon the observations of Axenfeld and of E. Fuchs, that an anterior choroidal detachment occurred after glaucoma iridectomies. Weekers, Krauss, Joudin, and Stoughtenborough found no communication between the anterior chamber and the suprachoroidal space; instead, they found a cicatricial adhesion of the ciliary body to the overlying sclera. In addition, Stoughtenborough failed to find an atrophy of the ciliary body at the site of the operation. This leaves us still at sea as to the modus operandi of this operation.

The second table will be a consideration of the accidents and infections which follow surgical intervention. The third will be a mention relative to sclerotomies, both anterior and posterior. The next will be a table of hospitalization based upon personal experiences.

The relationship of secondary glaucoma and of uveitis, of secondary glaucoma and of immature cataract, and of primary glaucoma with incipient cataract will be considered last. In regard to the last two above, two recent statements are of interest. While not new, their importance is too often forgotten. 1. Lancaster said, "They may be divided into two groups, (a) those where the cataract seems to have connection with the operation (of these the number is small), and (b) those where the cataract does not have connection with the operation (there are many of these because senile cataract is common at the ages when glaucoma is common). I think trephining and other operations which reduce tension tend to accelerate the progress of senile cataract." 2. Elliot, "I find postoperative cataract comparatively rare, altho (a) preexisting cataract may progress faster after the operation, or (b) a form of cataract secondary to iritis may develop sufficiently to call for later operation."

TABLE I.

Table of reoperations, of enucleations and of postoperative miotics.

A Reoperations.
B Enucleations.
C The continuation of miotics, after the operation.

	A	B	C
Trephining	Rare to occasional. From 2% to 10%. See (a).	Rare to less than occasional. 1 to 4 cases. See (b).	Ranged from none to all. The averages were from 15% to 35%. See (c).
Anterior sclerectomy	Rare to less than occasional. 1% to 5%. See (d).	5 cases reported. See (e).	From occasional to 80% of cases.
Iris inclusion operations	Occasional to less than frequent. The average was from 10% to 20%. See (f) and (g).	2 cases were reported. See (h).	Most cases to all cases was variously reported.
Iridectomy	Occasional to frequent. The averages reported were from 15% to 25%. See (i).	23 cases were reported. See (j).	From 50% of cases to all cases was variously reported.
Cyclodialysis	Occasional to more than frequent. The averages reported were from 8% to 50%. See (k).	None reported. See (l).	From 20% of cases to all cases was variously reported. See (m).

(a) It must be considered that this operation is applicable to all types of glaucoma. A trephine operation can be repeated. Elliot reported it rarely necessary.

(b) It must be considered that this operation is applicable to all types of glaucoma.

(c) Elliot reported them unnecessary; the operation was a failure if miotics were necessary. Massage is to be used when tension stays up before reoperation is done. We consider the need of miotics as an uncompleted case.

(d) Performed so much less frequently that this is an inadequate observation.

(e) Weeks reported but one in a series of 350 to 400 cases.

(f) Operation done an insufficient number of times for adequate observation.

(g) A difficulty with reoperations in this procedure is the fact that the maximum results are delayed after the operation. This can modify results when impatience is allowed to enter into the therapy. On the other hand, the ease of reoperation, using the same technic, is relevant.

(h) The factors which modify this are the ease of the operation, the ability to perform the same operation again, or even another type, the less severe postoperative reaction, the operation is not done in absolute glaucoma, and the fact that this procedure is not as common as are several others.

The accidents and other complications which occur in glaucoma surgery are largely connected, it seems, with sclerectomy and with trephining, because these two types of operations have a more complicated technic. The following, next table, will therefore include only these. They are not alone, however, in operative complications. A quiet iritis has been seen following iridotasis, especially in those cases where the iris inclusion is followed by a shower of uveal pigment into the anterior chamber. The accidents connected with cyclodialysis have been mentioned. The three instances of anterior chamber vitreous prolapse which occurred in that series could well be serious by reason of a further occlusion with vitreous of the filtration angle. The occasional partial denudation of Descemet's membrane may be uneventful, also somewhat less so. There are no statistics available on this, so it is open to as pessimistic a conjecture as any one may wish. Iridectomy is covered in the table, barring such accidents due to manipulations as acute and immediate infection. The author knows of two acute infections following iridectomy, in which the eye was lost. The first was a peripheral iridectomy for secondary glaucoma with iris bombé, the second was a chronic iridocyclitis following an acute infection in acute hemorrhagic glaucoma, with enucleation necessary later because of a painful sightless eye. We are concerned in this paper, however, with the late results and the late infections only.

(i) The facts which modify this are these: it seems to be the best for acute congestive glaucoma, it is often done with a deliberate plan for reoperation if necessary, and a trephining can be done after an iridectomy.

(j) So often done and for such varied indications, especially before 1910, the advent of the filtration cicatrix procedure. This frequent procedure, done under varied conditions and circumstances leaves a high incidence of painful sightless eyes, many in uveitis or postuveitis. See (i) above also.

(k) This is the easiest of all procedures to perform and to repeat in the same case, even to 2 and 3 times. It is done infrequently for congestive glaucoma, (the highest cause for reoperation), hence, with simple glaucoma, reoperations are not as commonly demanded. Often times used as a "court of last appeal," as well as for the control of glaucoma following cataract extraction. Elschnig, the greatest exponent and authority on this operation, reports 8% reoperations for the 2nd and 3d repetitions, a very low incidence, but he uses the operation only for glaucoma conpensatum (simple glaucoma).

(l) Points relevent here are the indications for the operation, the ease of reoperation, the fact that other operations can follow this procedure, as a trephining, and also that this operation is occasionally used after other different procedures, see (k) above, have failed.

(m) Elschnig who uses it wholly for simple glaucoma reported a 20% incidence wherein miotics are necessary. Gradle, in a series of 100 cases, reported the necessity for miotics in all cases following operation. This also is our experience.

TABLE II.

A Cataract by reason of manipulations.
B Late infections.
C Vitreous loss.

	A	B	C
Trephining	From 1 to 10 cases reported. Usual report was "rare." See (a).	21 cases were reported. See (b) for the number of eyes lost.	Very rare to never were the various reports. One case of author, one other known of. See (c).
Sclerectomy	Four per cent of cases. See (d).	Various reports. 1 in 350 cases. 1.3%, and 2.3% of iridocyclitis.	6.2% of cases. See (e).
Iridectomy	Occasionally a form secondary to iritis. See (f).	Vitreous loss and late infections not seen.	

(a) See Enroth's recent review, Acta Ophthal. 1927, v.5, pp. 113 to 121. Here again it seems as if that statement of Elliot's is lost track of, that "glaucoma occurs most commonly at the same age in which senile cataract is common, also that any operation for decreasing tension will hasten the maturation of an incipient senile cataract." Enroth reported 29% of cases following trephining develop cataract in the period studied, one month to 8 years. Of these, over 30% occurred in eyes with hypotony. (See Elliot's and Lancaster's quotations above.)

(b) Of this number, ten eyes were reported as lost, the remainder from "fair to poor vision." One case was reported with good vision.

(c) Author's case was bilateral glaucoma, right sightless and in absolute stage, left eye with tension over 60 mm. Hg. and with incipient cataract. Vitreous prolapses occurred when suture of flap was started. Later, lens extraction resulted in good recovery, tension 18 mm. Hg.

(d) Figures are Meller's and Wecks'.

(e) Figures are Meller's.

(f) Elliot's statement. This is wholly concurred in by the author.

TABLE III.

Table of sclerotomies.

	Posterior sclerotomy	Anterior sclerotomy
Recovery	Absolute glaucoma, no recovery of vision, but with normal tension occasional case (13 cases reported). One recovery reported following thrombosis of retinal vessel. No recoveries with simple or inflammatory cases.	No recoveries.
Adjunct treatment	Rather common prior to trephining in severe high tension, reported as being used preliminary to sclerectomy, preliminary to lens extraction with secondary glaucoma from lens swelling, and used preliminary to iridectomy for acute congestive and severe secondary with uveitis.	Reported variously as being used in from an occasional case to 20 cases.
Uveitis with iritic glaucoma	Not used save as mentioned immediately above.	Used from many times to very many times with good results. This seems to be a well established procedure.

TABLE IV.

Table of hospitalization and observation.

	Active hospitalization	Active observation
Trephining	10 days	1 month
Sclerectomy	14 days	1 month
Iridectomy	1 to 2 weeks	1 to 3 months
Cyclodialysis	6 days	6 weeks
Iris inclusions	6 days	1 to 3 months

There is a great time difference in the early results as seen in the filtration cicatrix operations (trephining and sclerectomy), as compared with the results of iridectomy and of iridotasis. In the latter, the maximum tension decrease is a late development as has been said before. The increased time necessary following iridectomy is not unusual, considering the many different causes for its single major indication, i. e., acute congestive glaucoma. The period of observation is in the nature of prophylaxis rather than of therapy.

TABLE V.

Table of complicated glaucomas.

A Primary Glaucoma with incipient cataract.
B Secondary glaucoma with immature cataract.
C Secondary glaucoma with uveitis (iritic glaucoma).
D Procedure most frequently utilized.

	A	B	C	D
Trephining, later extraction	40%			
Extraction with iridectomy and later trephining	18%			
Extraction with later a cyclodialysis	8%	12.5%		
Extraction preceded one-half hour by posterior sclerotomy	8%			
Acute glaucoma, a basal iridectomy	8%		20%	
Iridectomy usually, sometimes trephining	8%		20%	
Trephining below, later extraction above with iridectomy	8%	12.5%		
Totals with trephining as a basis				68%
Iridectomy with later extraction		60. %		
Sclerectomy with later extraction		12.5%		
Totals with iridectomy as a basis				60%
Paracentesis			30%	
Paracentesis with later iridectomy			20%	
Iris inclusion			10%	
Totals with paracentesis as a basis				50%

CONCLUSIONS.

It is proverbably a snare and a delusion to draw conclusions from statistics. On the other hand, they are our only basis for scientific deductions, as manifested by the very common tables promulgated by every insurance company, as an ordinary example.

Those which we may properly consider from the above are numerous, and unless controversy is desired (which is not invited), conclusions must be made with circumspection. These, however, do seem legitimate and logical.

1. Trephining is satisfactory for the largest number of cases of glaucoma, all types included, except that secondary to uveitis, and acute congestive glaucoma. See No. 3 below.

2. Anterior sclerectomy is limited by reason of its complications and contraindications.

3. While iridectomy is favored in many cases for acute congestive glaucoma, the above statistics show that trephining is also used at times, but only after the subsidence of the acute congestive attack, i. e., during a quiet interval (noncongestion). At this time, trephining, when done, is even more satisfactory than the results achieved thru iridectomy done earlier and during the congestive period. Unfortunately, a postponement is not always possible. Trephining during the acute phase is unsatisfactory,

hence iridectomy remains the operation of choice and of necessity for an acute congestive glaucoma.

4. Paracentesis is the basis for the treatment of iritic glaucoma, with the possibility of a subsequent iridectomy in the foreground.

5. Trephining with a later lens extraction is the logical operation for primary glaucoma complicated by incipient cataract.

6. An iridectomy with later extraction is the operation of choice for secondary glaucoma from lens swelling.

7. Cyclodialysis has a definite place in glaucoma surgery in the simple noninflammatory cases and as adjunct treatment following complicated glaucoma, but its incidence of recoveries is less than that of two other operations.

8. Iris inclusions operations call for a careful selection of the cases upon which it is to be practiced; the results are less dependable than are those from other procedures.

9. The technic of cyclodialysis and of iris inclusion, by reason of its simplicity, is of less danger to the eye in the hands of the occasional operator.

10. Posterior sclerotomy has a definite place in glaucoma surgery.

11. There is considerable chaos, at present, in the status of glaucoma surgery.

DISCUSSION.

Dr. Albert E. Bulson, Fort Wayne, Ind.: I have been requested by the essayist to compare the trephine and iridotasis operations, and if possible point out their comparative values. He already has argued the case for the trephine operation, and what I shall add, perhaps, will detract from that plea. In my discussion, I may analyse more fully some of the literature quoted by the essayist.

My first observation, based upon the study of the available literature, is that a number of excellent ophthalmologists have said that they have given trephining a good "try out" and then abandoned it. In this connection, it has been published that no less an authority than Fuchs,[3] while on a recent visit to this country, stated that he had done over one thousand trephine operations for glaucoma, but had abandoned the operation on account of the occurrence of late complications. In the same article,[1] the trephine operation was condemned by Zentmayer because iritis too frequently occurs, because secondary changes, usually the development of lenticular opacities, sometimes takes place, probably as the result of hypertonus, and because infections are relatively frequent. At the same meeting Crouch[1] said that his experience with the trephine operation has been "very unsatisfactory," an opinion also expressed by Hiram Woods.

Calhoun[2] says that he has seen every possible complication following the trephine operation, from delayed restoration of the anterior chamber and quiet iritis to a permanent detachment of the choroid, with blindness and late infections. He further said, he would not feel justified in recommending it or performing it upon his patients, because he would not choose to have it done on himself were his eyes in such condition as to require surgical treatment of glaucoma.

Davenport,[3] in considering the after results in 405 patients in Moorfields upon whom the trephine operation was performed, reported 80 per cent apparent successes, and 4.5 per cent complete failures. There were 14 cases of late infection, and quiet iritis was reported as a sequel in most all of the cases.

Wilmer,[4] in a series of 72 trephine cases, had 15.2 per cent failures, and in a second series of 35 trephine cases, he had 10.4 per cent failures. In the discussion of this paper, Whitehead said that he had trephined many cases, but now thought iridectomy safer. Wilder, in a discussion of the same paper,[4] reported having done more than one hundred iridotasis operations with but one failure, and said that he was glad that he had not elected to do a trephine on any of the cases. Gjessing, in discussing the same paper,[4] spoke unfavorably concerning the trephine operation, but reported 113 iridotasis operations, with 81 good and lasting results.

Gifford,[5] in comparing the results of trephining and iridotasis, says that with a well performed operation the results are practically identical.

In the proceedings of the Netherlands Ophthalmological Society,[6] it is reported that Tresling spoke unfavorably of trephine operations, and cited ultimate failures in cases at first counted as successes. Most of the other speakers at the meeting also spoke unfavorably of trephining.

Many more American and European ophthalmologists could be quoted as not satisfied with the trephine operation, and a good percentage of the number have admitted that they have abandoned the operation. On the other hand, a study of the available literature shows that all of the operators who have employed iridotasis enough to offer intelligent opinions concerning it, think highly of it. Those who have never used it, refuse *on theoretic grounds* to try it. I can find no single reporter who has used iridotasis and then abandoned it because the final results were unsatisfactory.

It is quite true that the value of any form of treatment must depend upon the end results in a large number of cases, observed over a considerable length of time. Furthermore, the immediate as well as ultimate effect of any operative procedures will depend in some measure upon the skill and technic employed by the operator, as well as the selection of cases. It may be granted, that some of the statistics presented in connection with both trephine and iridotasis operations include some cases that from their very nature would be considered almost hopeless under any treatment, and yet we have a right to assume that the results reported are a measure of the value of the operations under consideration.

Time does not permit reference to many reports concerning the iridotasis operation, tho I shall call attention to a few published statements, to some of which the essayist has called brief attention.

H. Galetski-Olin[7] analyzes 258 cases seen in Grönhohn's clinic during a period of fourteen years. There were 197 trephines, 50 sclerotomies, 20

iridencleises, and 18 iridotasis operations. They report that iridotasis appears to be the least difficult operation technically, and the least liable to postoperative complications. Further, the pressure is normalized most frequently after iridotasis, in 90 per cent of the cases, and central vision is best preserved after the operation. After trephine operation, the pressure is rendered normal much as after iridotasis, and oftentimes subnormal, but the technical difficulties are much greater, the liability to postoperative complications is more marked, and the visual results also are not quite so good.

Borthen,[8] reports 97 cases, more than a third of which had been under observation for two years or longer. With the exception of one case of chronic inflammatory glaucoma, in which iridotasis was ineffective, all these cases were cured, with resulting normal tension and vision, and fields as before the operation. All but the one case—chronic inflammatory glaucoma which was unimproved—were satisfactory. Of the entire series, it may be said that tension nearly always was reduced to normal, and as much vision was preserved as could have been saved under any conditions.

Harrower[9], who is credited with having introduced iridotasis in this country, reported twenty-three operations, all satisfactory, some of which had been observed over a period of five years, with vision, fields, and tension unimpaired. Still later, he added 52 to the series without changing his opinion of the end results.

Goldenberg[10], in a general summary of results after five years of experience with iridotasis in all forms of glaucoma, says that the operated cases disclose "a distinct freedom from all symptoms of increased tension." In a later contribution[11], he reiterates his faith in iridotasis, and says further that "if a glaucoma operation is indicated, in the presence of a mature or immature cataract, there would seem to be no contraindication to the iridotasis technic." As a comment on how iridotasis becomes effectual, he says that "it still is an open question whether the benefits derived from the iridotasis operations are wholly due to the seepage or filtration at the point of incarceration, or to the traction on the iris upward which frees the more natural channels of escape."

Wilder[12], in a series of forty-eight cases operated by iridotasis, reports success in 90 per cent of the cases, or as good as in trephining or other operations, and with much less risk. At the Oxford Ophthalmological Congress in 1927, he[13] again spoke in favor of iridotasis, and said that in over one hundred cases operated, he had encountered but one failure, and that one might have occurred after any operation. Some of his cases were operated two to twelve years ago, and he said he is thankful that he decided to do iridotasis rather than trephining. At the same Congress, Gjessing[13], of Norway, spoke of 113 patients upon whom he had done iridotasis, with 81 cases of good and lasting results, in a period of seven months to eleven years after operation. Even Wilmer[14], who admits not doing the iridotasis operation, on theoretic grounds, includes in his statistics of operative results for glaucoma 517 iridotasis operations, by various surgeons, with 87.4 per cent successes, which is a better average by 7 per cent than that obtained in a still large number of cases in which trephining was done.

Gifford[15], in comparing the results of trephining and iridotasis, reports 82 iridotasis operations with but one primary infection, and asserts that "with a well performed operation the results are practically identical."

Holzer[16], sent a questionnaire to ophthalmologists thruout the country and out of 307 replies, only 16 favored iridotasis, but it will be noted that most of those who answered the questionnaire admitted not having had any experience with iridotasis. In choosing an operation for themselves, if needed, 17 stated that they would select iridotasis. Holzer also mentions a personal communication from Harrower, who introduced iridotasis in America, to the effect, that in none of his 75 cases, has he (Harrower) had late infection.

Durr[17] quotes Holth as saying that late infection occurs in 7 per cent of all trephined eyes, but gives no unfavorable reports concerning iridotasis. He credits Allport with 33 cases, Harrower with 75 cases, Stieren with 6 cases, and Darling with an indefinite number, all favoring iridotasis.

Verhoeff[18] says he has found iridotasis superior to the trephine operation in cases of chronic congestive glaucoma, because in such cases a trephine opening is especially apt to become closed by the tissue reaction.

Dupuy[19] credits John Dunn of the Charity Hospital in New Orleans as the first man in New Orleans to practice iridotasis on a large scale, and with such marked success, that the author began to use the procedure in any glaucoma case thought requiring operation. In a total of over 100 cases, Dupuy says the results have been lowered tension, pain relieved, and vision and fields retained. In none of the cases were there any late infections.

My personal experience with iridotasis has been instructive to me. Three iridotasis operations were done in 1927, with immediate good results, but following the general unfavorable criticism of the operation on theoretic grounds about that time, I abandoned the operation until a few years later, when favorable reports concerning the success of the operation began to appear in the literature, and I had discovered that my operations done in 1927 still remained successes, in that tension remained reduced, fields were stationary, and vision continued good. I also had discovered that my trephine cases had not been generally successful. I have no regrets in resuming the employment of the iridotasis operation, for while I have had one complete failure, due to an imperfect operation, the results in general have been very satisfactory.

The final analysis of the comparative results of all operations for glaucoma must depend upon end results in a large series of cases observed over a long period of time. Even then, the more favorable results will depend upon the skill of the operators, the technic used, and the character of the cases operated. Iridotasis is not difficult to perform, but it does require a careful technic, if the best results are to be secured. It is adapted to any form of glaucoma, but like trephining or any other operation, it is more successful in some cases than others. It does have the advantage of drawing the iris away from the filtration angle, while at the same time creating an avenue for filtration. Up to the present time, there are few cases of late infections reported, and even the question of doing the operation in the presence of incipient cataract has been

answered favorably. No cases of hypotension or of sympathetic ophthalmia have been reported.

Finally, the general acceptance of any operation as the operation of choice for glaucoma must take into consideration its adaptibility to the average operator as well as to operators of great skill and extended experience in all operative procedures. This means, that the operation must be easy of performance, free from technical difficulties, with a minimum of risk during or after the operation, and with not only satisfactory but lasting results. The experiences of many operators who have been quoted seem to indicate that in iridotasis we have found such an operation.

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Dr. E. C. Ellett, Memphis, Tenn.: This interesting paper attempts to crystallize as nearly as can be the opinion and practice of our day in regard to this important question of the treatment of glaucoma. Without meaning to minimize the importance of the many other matters discussed in the paper, the little time allowed for discussion will be spent on certain facts in Table 1.

Of the five operations considered in that table, sclerectomy and cyclodialysis are not in sufficient favor to be seriously considered in the average case, by the average man, tho cyclodialysis has a few warm advocates and fulfills extremely well the indications in a small number of cases. The fact that the author has found that "most to all" cases of iris inclusion operations required the continued use of myotics, would indicate that it is hardly a curative procedure, tho in the cases reported by Wilder in 1923, none required myotics after the operation, and in 90 per cent of these cases tension, was permanently reduced. That the good result in such cases may be delayed, as is pointed out, was well illustrated in one of my own cases, in which the operation of iridotasis was performed in July, 1921, and it was eighteen months after that, namely, March, 1923,

before all treatment stopped. Since that time, now over five years, the tension has remained low and the eye is apparently cured of the glaucoma.

Interest centers, and I feel sure the discussion will center, in the relative advantages of the sclerocorneal trephine and iridectomy, and in most cases it will consist in a relation of personal experiences. In my own small experience, the preference was for a time for the Lagrange operation in chronic glaucoma, and for iridectomy for acute. The large wound of the Lagrange operation does not add to one's peace of mind, tho the results were quite good, and there are still some eyes under observation and in excellent condition after as much as twelve years from the time of operation. Iridectomy was, and in most quarters still is, the operation of choice for acute glaucoma, but it has always been a technically difficult and to me a surgically unsatisfactory job. My own cases and those of others that I have seen, almost never present the hoped for appearance of a wide and deep coloboma with free pillars, and while we operate to preserve sight and not for appearance's sake, nevertheless, we like to turn out a piece of work that we are willing for our most envious colleague to see. It gradually dawned on me that the trephine cases most nearly met this situation, with at least no more failures or surgical misfits than any other operation. An iridectomy on an inflamed and tender eye, with a nervous and excited and worried patient, a vascular iris, a shallow anterior chamber, and a few other difficulties that need not be recited, is about the hardest task I know, tho I also know about morphin and hyoscin, and the various tricks of local anesthesia, as well as the possibility of general anesthesia. But as far as my own experience goes, I have found that a better, broader, deeper and more regular iridectomy, with less danger of wounding the lens, loss of vitreous and other disasters, and with all the advantages of any iridectomy, can usually be obtained with the sclerocorneal trephine rather than with the usual technic of an iridectomy. So that at this time, I think Elliot's advice to trephine all cases fits in with my own limited experience. This is not intended as a debate, and I am not anxious to argue this matter, but to give my own idea at this time. The trephine operation is easy to repeat, and the small bulbar opening helps to a rapid convalescence. It has been my good fortune not to have seen an instance of late infection of a trephined eye, and when I do, I may not be so much in favor of the operation.

It is pitiful to think how little we know about glaucoma. We are realizing every day that we have underestimated its frequency, and are continually surprised to see in how many cases of cataract extraction, iritis and uveitis, as well as corneal disease, glaucoma represents the final, fatal stage. It is worth all the time we spend in investigating and studying and fighting it, and such a paper as this should be presented frequently, to enable us to compare notes on the disease and to confer as to the best means for its management. At present, we have to be content to fight one symptom, the increase of intraocular tension.

Dr. E. E. Blaauw, Buffalo, N. Y.: The presentation of this subject by Dr. Spaeth is a master stroke. Dr. Spaeth is a perfect operator, and in talking this thing over with him, we agreed that there are men who do not operate as much as Dr. Spaeth does, who do not do it easily. For myself, I think another operation is less difficult and disturbs

the eye less. In my opinion, the going in from the outside makes things a good deal easier. I have done a very limited number of these cases. Most of us do not operate every day.

I have heard that Dr. Spaeth is interested in the question of light sense, but that we may not speak about. But he is going to give us a little instrument, and show us the lines of light sense in glaucoma.

Dr. Allen Greenwood, Boston, Mass.: In 1910, I published a paper (Jr. A. M. A., July 16, 1910, vol. LV, pp. 190-194) on the Lagrange operation, giving the results in eight cases. Later I reported 25 cases. Some of those cases I have since followed for many years, and the operation has proven very useful. After Col. Elliot came to this country, I adopted the trephine operation and had one unsatisfactory result. After I had done a number, I operated on an elderly lady one April and got a good result, but in October of the same year, she had an acute conjunctivitis and lost the eye from panophthalmitis.

I still use the trephine operation, but the last few years I have done an operation where I combine a Lagrange with the iridencleisis operation. I make a conjunctival flap first, then an incision far back, and then with the Lagrange punch, I remove a bite of the beveled sclera. All the Lagrange operations I have done so far have given me a good filtering scar.

Dr. Harold Gifford, Omaha, Neb.: With regard to the way in which iridotasis works, several have spoken about the effect of iridotasis being due to the pulling forward of the iris and thus liberating the angle. This is what happens. When you pull a piece of iris out in the limbus and leave it there, it does not free the angle. On the contrary, it blocks it that much more. What it does do, is to turn the piece of iris inside out, so that you have a tissue covered on one side with epithelium, which does not adhere to the edge of the incision in the limbus.

Dr. Knapp, in his talk this morning, very aptly made the point that the danger zone for later infection is the place where you split the cornea. You cannot get your trephine far enough down into the cornea in most cases without splitting the cornea, and when you do that you have a very delicate layer of tissue just over the hole, thru which infection easily penetrates into the globe. I had one case, in which I did a trephining and got a beautiful result. The man came back in six weeks with an infection starting at the point where the cornea had been split, and he lost his eye inside of two or three days.

As to what can be done to prevent late infection, any man who has a thin flap following the operation has a sword hanging over his head. He should be told that this flap, altho it is a safety valve, is also a danger point, and he ought to do everything possible to keep it from getting infected. I have every one of these patients use a solution of chlorid of zinc (1 grain to the ounce), or any antiseptic that does not stain the eye, and it should be used indefinitely—the remainder of their lives. That, in most cases, will keep them from late infection. Tell these patients, that if there is the slightest sign of infection or irritation, they should hurry to the nearest oculist, and if that is not possible, should use hot applications freely and the zinc solution several times a day, till help can be reached.

DR. S. B. MUNCASTER, Washington, D. C.: I wish to speak particularly about the incision in operating for cataracts, where a previous operation for glaucoma has been performed by Elliot's method. At different times, I have made three kinds of incisions, the first being by cutting thru the cornea, the second by making a sclerocorneal incision thru the bleb, and the third by dissecting a crescent shaped conjunctival flap from above down to the bleb, and then making a sclerocorneal incision thru the bleb and under the dissected flap. I have found the latter method best, because cloudiness of the cornea is prevented by the incision passing at the sclerocorneal junction. Vitreous is usually prevented from escaping, and if vitreous should appear, a quick closure can be made, since I keep a suture in place connecting the flap and conjunctiva above.

DR. HARRY S. GRADLE, Chicago, Ill.: I wish to speak of one point, namely, cyclodialysis. A great many operators have reported results from cyclodialysis that are not comparable with results obtained with other operations for glaucoma. I believe the major part of these results is due to the fact that there was a poor indication for operation. Cyclodialysis is not a universal cureall; it is not indicated in every type of glaucoma, but there are certain definite indications for the use of cyclodialysis and if these indications are followed carefully, the end results will be more than satisfactory. What are these indications? I believe that the original Heinie dictum still holds true. The more urgent the indication for operation in glaucoma, the less beneficial are the results from cyclodialysis, and the less urgent the indications for operation, the better are the results from cyclodialysis. What does that mean? It simply means that in inflammatory glaucoma of the noncompensated type, cyclodialysis is not indicated. It means that in glaucoma simplex, the noninflammatory type, cyclodialysis is indicated, but in the severe, malignant type, it does not yield good results, and they should not be operated upon by cyclodialysis. But the cases just over the border line that are not controlled by miotics give exceedingly good results when cyclodialysis is performed. As I said, the operation is not a cureall; it is not desirable for operation in every type of glaucoma, and it is manifestly unfair to the operation to give results when it is used under conditions for which it is not indicated.

LIGHT THERAPY IN OPHTHALMOLOGY.*

S. R. GIFFORD, M.D., F.A.C.S.

OMAHA, NEB.

The importance of light in the life processes of plants and animals is too well known to require emphasis. The exact mechanism of its effect upon living tissues is by no means completely understood, but we know certain phenomena which are produced by light of different wave lengths. The spectrum may be divided into the infrared rays, of wave length from 20,000 to 8,000 Angström units, the visible rays, from red at 8,000 to violet at 4,000, and the ultraviolet from 4,000 to 1,000 Angström units. In this region, it is chiefly the rays below 3,000 which give the desired therapeutic effect. Below the ultraviolet are the X-rays and the shorter rays of radium. The infrared and visible rays are absorbed by the tissues and their energy is converted to heat, producing dilatation of the capillaries, exudation of serum into the tissues and an acceleration of the local biochemical processes. The shorter ultraviolet rays are nearly all absorbed by the cells of the superficial layers, producing an abiotic reaction, the manifestations of which are a hyperemia, death, and desquamation of the most superficial layers, and an increase in the bactericidal power of the blood.

Duke-Elder[1] has suggested an ingenious explanation of the difference between the reaction of tissues to heat and the abiotic reaction produced by ultraviolet light, which is in line with modern theories of atomic physics. He believes heat or infrared rays are absorbed only by tissues whose atoms have a periodic movement corresponding to the wave length of these rays, and this absorption produces an acceleration of atomic movement. The shorter ultraviolet rays find corresponding frequencies only in the intraatomic movements of the electrons, and are absorbed by them, changing their orbits in the atoms, and so affecting the tissues in a more vital way. Unicellular organisms are most markedly affected by ultraviolet light. Planck[2] states that B. Coli

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is killed by an exposure of one to 20 seconds to light of wave length 2,200 to 3,050 Angström units. Staphylococci are killed in ten to sixty seconds, tubercle bacilli in twelve to thirty seconds.

The most familiar example of this abiotic reaction in the tissues is, of course, sunburn. While this occurs after any sufficiently long exposure to strong sunlight, it is especially common and severe on exposure in high altitudes, or where the sun's rays are reflected from water or snow. This is because the atmosphere at low altitudes absorbs a large part of the sun's ultraviolet rays, while sunlight at high altitudes contains a much larger proportion of ultraviolet, and that which is reflected from snow or water is broken into light of shorter wave length.

When the cornea is exposed to a source of ultraviolet light of sufficient strength, an abiotic reaction begins after six to eight hours, with swelling and irregularity of the corneal epithelium, with the formation of vesicles. If the exposure is longer, the epithelium is completely desquamated, the corneal corpuscles show fragmentation of their nuclei, and eosinophil cells appear in the cornea. This occurs, of course, only with exposures much longer than those used for therapeutic purposes. The same effect may be produced on the lens capsule, and opacity of the lens fibers may result from larger dosage, but such abiotic reactions are not seen in the lens in practice, because too much of the ultraviolet radiation is absorbed by the cornea and aqueous.

In considering phototherapy, it is important to know the range of absorption of different forms of light by the media of the eye, as it is only the light which is absorbed which is effective, or, in larger doses, harmful. Numerous investigations have recently been made on this subject, and the figures of Duke-Elder may be taken as substantially correct. The normal cornea absorbs 50% of the light from 18,000 to 16,000, the other 50% being absorbed chiefly by the lens. Between 16,000 and 13,500, the cornea absorbs almost all the light, while from 13,500 down to the beginning of the visible red rays at 7,700, an increasing amount of light is transmitted, amounting to 100% for the rays of the visible spectrum. In the ultraviolet region it absorbs practically all rays below 2,950.

The aqueous and vitreous transmit all the rays which pass thru the cornea. The lens, however, besides absorbing most of the longer infrared rays which pass the cornea, also absorbs 25% of the rays between 13,500 and 11,000, all the ultraviolet rays between 2,950 and 3,200, and part of those between 3,200 and

4,000. Thus, by the cornea and lens, the retina is protected completely for all practical purposes against an abiotic reaction from ultraviolet light. The damage which does undoubtedly occur to the retina after looking at an eclipse of the sun, and perhaps in snowblindness, is due to a concentration of infrared rays by the dioptric media, but not to ultraviolet light. While it is possible that repeated doses of ultraviolet light might have a cumulative effect on the retina by means of the longer rays between 3,200 and 4,000, this is unlikely. The retinal changes described by Birch-Hirschfeld in rabbits were chiefly in those from which the lens had been removed, and after exposures much longer than those used in treatment. Chotzen and Kuznitzky, after exposures of fifteen minutes to the Kromayer light, a dosage sufficient to cause severe corneal reaction, saw no changes in the lens or retina of animals or patients, and Koeppe treated several aphakic patients over long periods without seeing any retinal changes. Scheerer's two cases are the only ones of which I could find record, which developed cataract apparently as the result of intensive light treatment, and in these cases some X-ray treatments had also been given.

The chance of a harmful effect on the lens from infrared rays, and even from ultraviolet rays, must be seriously considered, and here the form of light used is very important. The mercury vapor arc, for instance, is rich in ultraviolet rays, some as short as 2,000 Angstöm units, and produces an intense abiotic reaction, while, being poor in infrared rays, involves no great danger to the lens. The carbon arc, used in the Birch-Hirschfeld irradiation lamp, is rich in both kinds of light, so filters must be used with it to cut off the infrared rays. Ordinary incandescent lamps are not suitable for ultraviolet radiation, since their glass bulbs absorb most of the ultraviolet rays. Quartz, however, transmits most of the ultraviolet, while special kinds of glass transmit almost as much. Uviol glass transmits rays down to 2,900, while absorbing a large amount of the infrared rays. Helio glass, a new clear glass made in America, is said also to transmit rays down to 2,900.

The Curay light makes use of an incandescent lamp whose bulb is of a special kind of glass, which transmits some ultraviolet. Spectrophotographs, however, show that very little light below 3,000 A.U. is emitted by this lamp. (Fig. 1.) For general irradiation of the body, the larger mercury vapor arc lamp is usually used, in a quartz tube. Types of this are the Jesionek

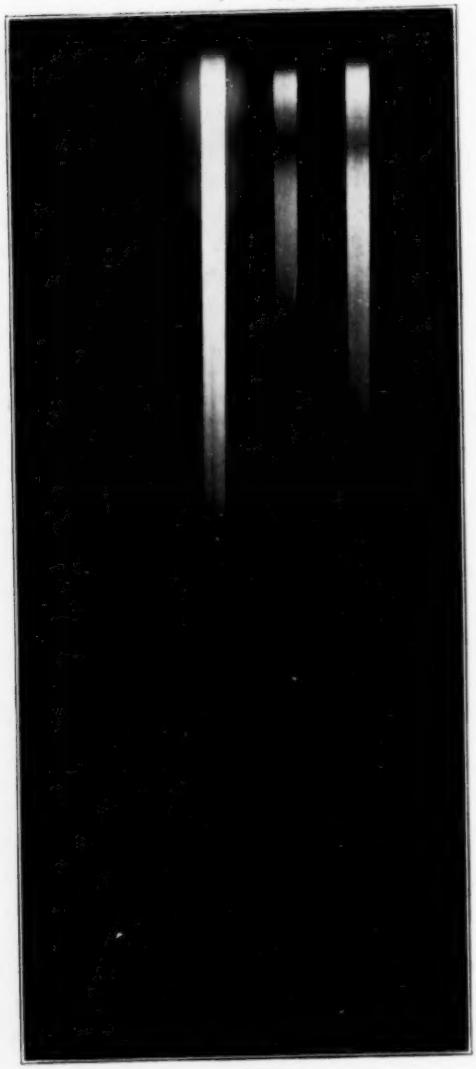

Figure 1.
Spectrophotograph of tungsten incandescent lamp with quartz applicator. (Curay light).

light, a light described by Duke-Elder[6] which, with the addition of a screen and quartz applicator, is also suitable for local applications to the eye, and the Alpine Sun lamps, now being used rather extensively, especially by pediatricians, and which Koeppe has used. In Copenhagen, where the Finsen iron light was first used for tuberculosis, a type of large carbon arc has finally been selected as the one whose spectrum most nearly approaches that of the sun, and this is being used for general irradiation.[7] The object of this treatment is to produce a mild skin reaction, which has been shown to increase the bactericidal power of the blood, and so is of value especially in tuberculous conditions and phlyctenular keratitis. In the technic used at Moorfields Hospital, one-third of the body surface is irradiated at each sitting with the mercury arc lamp, the dose being found which will produce a slight erythema, and this is repeated on other areas two to three times a week. After fifteen such treatments, a carbon arc light is then used which is richer in the long ultraviolet rays, and has a more penetrating effect. Five treatments with this are given, and after two to three weeks of rest, another course is given if necessary.

For local treatments to the eye with ultraviolet light, Duke-Elder uses a mercury vapor arc slitlamp apparatus, with a quartz lens system, Koeppe the Kromayer lamp, a small mercury arc in a water cooled quartz container, with a quartz applicator. The light used in most European clinics, and which I have used in most cases, is the Birch-Hirschfeld irradiation lamp, a carbon arc, with a filter of uviol glass, and a quartz lens system, one lens of which is movable, so that a fine bundle of light can be focused on the corneal lesion. (Fig. 2.) A quartz cell containing 5% copper sulphat solution is used to filter out the heat rays, but the uviol filter also removes a large proportion of these, so that without this filter only a very slight heat effect is produced, which I have never found harmful, and which may have advantages. The uviol filter removes most of the visible rays which would produce dazzling, transmitting only a narrow band in the blue-violet, the effect of which is rather to decrease photophobia and corneal sensitivity. Figure 3 shows the spectrum of this arc light. Without filters, it transmits a uniform band extending down to 2,300 A. U. With the uviol filter, the shorter wave lengths are cut off, but a considerable amount of the ultraviolet down to 2,850 A. U. is transmitted. With the uviol and copper sulphat filters, however, the band stops at 3,140 A.U. so that

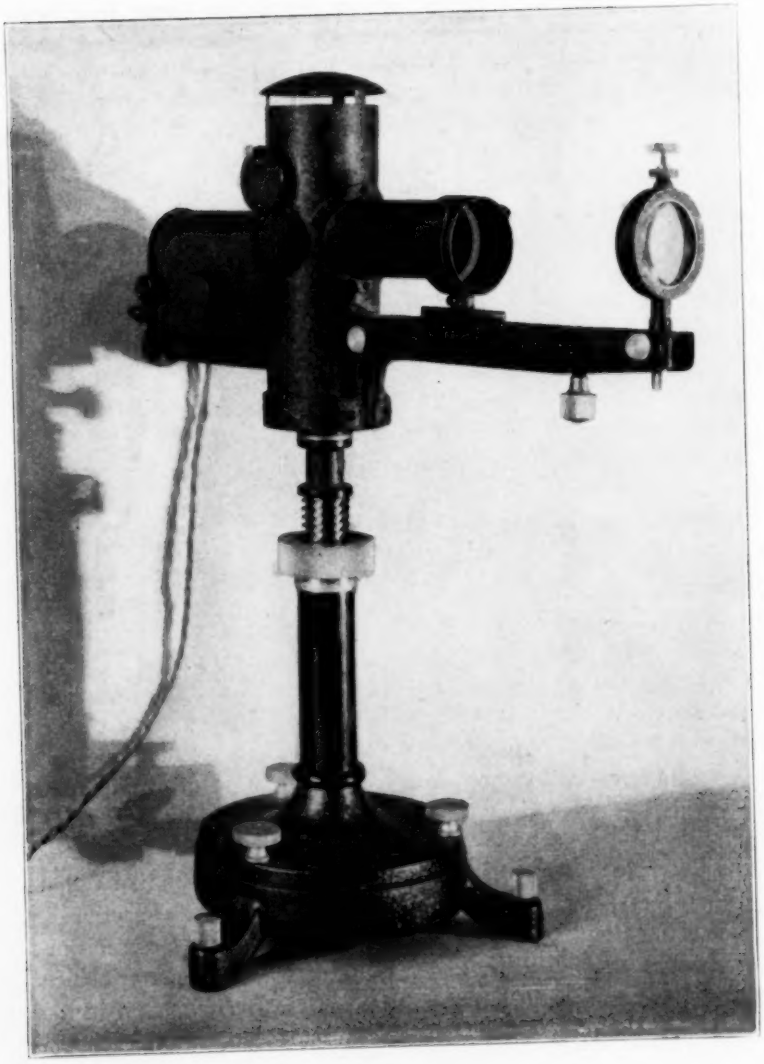

Figure 2.

practically no effective ultraviolet is transmitted. (Fig. 3.) Since the heat effect without the copper sulphat filter has not produced any harmful effects, in my experience, it would seem best to use the light without it—necessary, in fact, if any benefit from the ultraviolet is to be expected. Other filters, which transmit more ultraviolet than the uviol filter, may prove more suitable than uviol glass, and some attempts are being made with the assistance of Dr. F. Lowell Dunn of the Department of Experimental Medicine, University of Nebraska Medical College, to find a system of filtering which will be safe and with which the amount of ultraviolet can be varied satisfactorily. The amount of ultraviolet emitted by the light itself is much affected by the carbons themselves, carbons cored with nickel, aluminum, cobalt or iron being especially rich in ultraviolet.[8] In the arc used in this work, carbons with iron cores were used. With this apparatus, as at present used, the light is focussed on the lesion for five to ten minutes, and should be kept in focus by the surgeon or a trained assistant, as slight movements of the head or eye will otherwise remove the lesion from the area of effective radiation. Treatments are repeated once or twice a day.

Another method of phototherapy utilizes chiefly visible rays, which penetrate the media and are effective in lesions of the posterior segment. For this, the arc light, with Henker's filters of crystal violet and copper sulphat solution, is used. Koeppe reports good results from this method in 200 cases of tuberculous iridocyclitis, and in several cases of periphlebitis of the retinal vessels and choroiditis.

I have had no experience with this method of treating deeper lesions, and believe the evidence in favor of its use is not nearly so convincing as that in favor of ultraviolet irradiation in the types of condition to be described.

Phototherapy was first used for the treatment of tuberculous eye diseases and phlyctenular keratitis in scrofulous children, following the work of Rollier and of Fleming and Krusius on exposure to sunlight in high altitudes. In these conditions, general light baths with the mercury vapor arc have proven of definite value. Stock,[9] Koeppe, Duke-Elder and many others have reported rapid clearing up of eye lesions during the general reaction so produced, so that light baths are now a part of the routine treatment of phlyctenular keratitis in many clinics.

In tuberculous iridocyclitis and sclerosing keratitis, supposed in most cases to be due to tuberculosis, local applications of

A—Carbon arc without filter.
B—Carbon arc with uviol filter and 5% copper sulphat solution in quartz cell.
C—Carbon arc with uviol filter.

Figure 3

Spectrophotograph of Birch-Hirschfeld irradiation lamp.
Time in all—5 seconds

light have also been used. Koeppe's 200 cases of iridocyclitis which have been mentioned, were treated with an apparatus which depended chiefly on the visible rays. Schanz[10] used his arc light with uviol filter for similar conditions. Both Stock and Duke-Elder, however, report that no definite results are obtained by local phototherapy in tuberculous iridocylitis, altho Duke-Elder believes a trial of such treatment is justified where tubercles are confined to the iris. Stock and Gilbert[11] have seen good results in sclerosing keratitis.

Lupus of the lids and conjunctiva was first treated by Lundsgaard[12] with the Finsen light, which he found superior for this purpose to the mercury vapor lamps because its spectrum contains more of the longer ultraviolet rays, so that a deeper effect is produced. The cornea was protected by cotton and the light applied to the everted lids, which were made anemic by pressure with a quartz applicator. Treatment lasted one to two hours, producing a second degree erythema with membrane formation. The Kromayer light requires much shorter exposures, and good results have also been obtained with it. Axmann[13] used a mercury vapor arc with success in similar cases. He did not protect the cornea, and saw corneal opacities clear up under the irradiation.

Lundsgaard and Grönholm[14] submitted 109 eyes with trachoma to treatments with the Finsen light by much the same technic. They report that many were cured after one or two applications, old cases with the disease deep in the lids being among those cured. In some cases, more treatments, as many as seven, were necessary. Isaak[15] treated 22 eyes in the same way with good results, the follicles often disappearing in one week. Mohr and Baum[16] and Chotzen and Kuzintzky used the Kromayer light with a special quartz applicator the shape of the lid, backed with metal so that the cornea was protected. In early cases, the results were much better than by the ordinary treatment, but in cases with extensive scar formation the effect was not deep enough. Marked clearing of the pannus was seen in many cases. Lundsgaard later (1918) reports that with the Finsen light, the length of treatments need only be twenty to thirty minutes, and that two treatments ofen produced healing. Duke-Elder reports good results in vernal catarrh with his mercury arc slitlamp.

The treatment of serpent ulcers by light was begun by Hertel,[17] who used a magnesium light in 26 cases. Two-thirds

of these cases healed with much slighter scarring than was shown in similar cases treated by other methods. Schanz, in 1913, reported on the use of a carbon arc and also the quartz lamp, both of which delivered light of short enough wave length to kill bacteria in the cornea. While his results were good, he did not believe they surpassed those obtained by other methods, and gave up this form of treatment in favor of optochin. His arc light with uviol filter and quartz lenses was in its essentials much the same as that used by Birch-Hirschfeld. This author[18] has been the most enthusiastic advocate of phototherapy for serpent ulcer, and considers it the treatment of choice, as bringing about healing in the shortest time with the least scar formation. Up to 1924, he reported 280 cases treated in the Königsberg Clinic, with 8.5% of eyes lost. Hoffman[19] reports on 190 cases treated since then, with only 4.6% losses. Before the use of phototherapy, the losses averaged 32%. The arc light with uviol filter and quartz lenses was used for treatments of 4-10 minutes, given once or twice a day. Fluorescein or rose bengal, 2%, is first instilled into the sac to stain the cornea, as these dyes are believed to sensitize bacteria to the action of light. Birch-Hirschfeld advises using rose bengal for the first treatments, as having most effect on the organisms, then fluorescein, and finally, when there is delay in epithelization, rose bengal and short applications of light (3 minutes). Some interesting experimental work has been done by Passow[20] on these "sensitizers." Exposing plates inoculated with staphylococcus aureus to the arc light, he found that without sensitizers, 120 seconds were required to kill the organisms, while when fluorescein was first used, the organisms were killed in 30 seconds, and when 2% rose bengal was used, in 10 seconds. Using a Hammer lamp which transmits chiefly visible rays, and rose bengal, the organisms were killed in 10 seconds, while without the rose bengal this light was without any effect. Much the same results were obtained when organisms were exposed in fluid of various depths, the Hammer lamp and rose bengal being the only combination which could kill organisms in layers .8 mm. deep. In pus the arc light with rose bengal was effective only in layers .1mm. thick. In experimental corneal ulcers produced by B. suisepticus, irradiation with the Hammer lamp and rose bengal produced better results than the other methods which were tried, and seemed to justify the belief that such treatment in serpent ulcer could have a bactericidal effect. Planck states that sensitizers increase the bactericidal

effect of visible rays, but have no effect on ultraviolet rays, which seems to be confirmed by Passow's results, which apparently depended chiefly on the visible rays. Gilbert reports excellent results in serpent ulcer with the Birch-Hirschfeld lamp, which has made paracentesis and the cautery unnecessary in his cases. Stock, however, who has tried the same light in various conditions, reports no good results in serpent ulcer. Passow's demonstration of the enormous effect of a very thin opaque layer of pus in diminishing the effect of light, would suggest that a bactericidal effect would be very difficult to obtain in a serpent ulcer when the bacteria were lodged deeply in the partially opaque cornea and were covered by a layer of exudate.

In herpetic keratitis, including dendritic ulcer and allied conditions, however, the virus affects the superficial layers and here, as would be expected, good results are reported by phototherapy. Stock reports almost unfailing success in these conditions, one treatment of 5 minutes often being sufficient. Peppmüller[21] reports good results in severe cases. Of other forms of keratitis, phlyctenular keratitis has not usually shown a favorable response to local light treatments, while it does usually respond to general treatments. Interstitial keratitis has not usually responded well, altho Hensen reports good results in the avascular form by the use of the Alpine sun lamp locally for short exposures (¼ to 1 minute). Tubercular keratitis has done well under the treatment in the hands of Chotzen and Kuznitzky and others. Stock reports one good result in rodent ulcer.

Blepharitis which does not respond to the usual methods has been treated by Duke-Elder with the mercury vapor slitlamp with good results, and by Poyales[23] with the carbon arc light. I have used general light baths in severe cases of phlyctenulosis, sclerosing keratitis, tuberculous iridocyclitis and iridocyclitis of unknown origin. I believe they have been of distinct value in clearing up these obstinate conditions, and have been encouraged to make use of them more frequently. They are now being used in all cases of phlyctenulosis at the University Hospital. Like foreign protein injections, light baths often seem to produce a sudden shower of antibodies in the blood, sufficient to start the healing process. Naturally other methods of treatment are not neglected in these cases.

My experience with local phototherapy is that derived first from a series of 53 cases in which this method was carried out more or less consistently. The apparatus used in most cases

was the Birch-Hirschfeld carbon arc light with uviol filter and quartz lenses. In a few cases, especially those of blepharitis, the Curay light with quartz applicator was used. The conditions treated were: herpetic keratitis, including dendritic ulcer, 15 cases; epithelial dystrophy and recurrent erosion of the cornea, 9 cases; deep keratitis, including sclerosing keratitis, 6 cases; iritis, 1 case; blepharitis, 6 cases, trachomatous corneal ulcer, 5 cases; other types of corneal ulcer, including serpent ulcer, 5 cases; burn of the cornea, 2 cases; gold chlorid staining of the cornea (to reduce the gold), 1 case; phlyctenular ulcer, 2 cases; Mooren's ulcer, 1 case.

The fallacies of judging any method of treatment applied to such a series of cases, in no two of which were the original lesions identical, are apparent. An accurate comparison of treated and untreated cases can not be made outside the laboratory. In these cases, additional therapeutic measures were often used, so that none of the results obtained can be ascribed to phototherapy alone. All that can be done is to compare one's recollection of the course of such cases with and without phototherapy, as regards pain, length of time required for healing, and visual disability produced.

In the first place, any claim that phototherapy is a specific treatment for serpent ulcer has received no support from this series of cases. The few serpent ulcers which were given phototherapy were also given the usual forms of treatment, applications of optochin and trichloracetic acid and delimiting keratotomy. Their course differed in no way from that of other serpent ulcers, except that the pain and photophobia were usually relieved for some time following each treatment. In spite of the enthusiastic reports of Birch-Hirschfeld and others, the treatment was not begun with much confidence of success, because of theoretic considerations. Where, as in serpent ulcer, the bacteria are located deep in the cornea and separated from the surface by a layer of exudate and opaque cornea one-half to one millimeter thick, no great amount of ultraviolet light can reach them, probably not enough for a bactericidal effect, even when "sensitization" by various stains is employed. In very early serpent ulcers, none of which were seen in this series, phototherapy might be effective, and as Birch-Hirschfeld has claimed, with a minimum of damage to the cornea.

The most marked effect of phototherapy was seen, as might be expected, in superficial corneal lesions, such as herpetic or

dendritic ulcers, which tend to spread over much of the cornea without involving the deeper layers. Here treatment by strong caustics or by paracentesis has, in my experience, been harmful, as apparently offering a foothold for the herpes virus on necrotic tissue, and allowing a deeper involvement of the cauterized areas. Fifteen such cases were given phototherapy, an exposure of three to five minutes being given every two days, till the ulcer ceased to stain with fluorescein. Rose bengal or fluorescein was used before treatment in all cases. The number of treatments required varied from four or five in mild cases to twenty-five in one case which showed several recurrences. All cases healed with a thin scar allowing useful vision, and altho there were recurrences in several, these responded to the same treatment. In most cases, the time required for healings was considerably shorter than that which would have been expected from the type of ulcer present. The most marked effect was seen after the first few treatments, and in five cases healing occurred after two to four treatments, with almost negligible scar formation. (In one case, this occurred after one treatment.) Later treatments seemed to have less effect. While this may be explained by the assumption that the herpes virus was deeper seated in these cases, a distinct impression was formed that the cornea became tolerant of the light and ceased to be stimulated by it, or that the resistance of the virus to light was increased. After stopping treatment for varying lengths of time, however, and then trying it again, as in the cases with recurrences, the original effect seemed to be obtained. One interesting case of herpetic ulceration following a malarial inoculation healed rapidly several times after two to three treatments, but showed a tendency to break down after going without treatment for one or more weeks. The eye was free from irritation when last seen, in spite of staining slightly, and as vision was not disturbed, the patient did not return for further treatments.

A few cases where other forms of treatment have been tried without effect seemed to show most definitely the value of phototherapy. A man of seventy-two had been treated elsewhere for corneal ulcer for the past three weeks, without relief. After coming into our hands, the ulcer, a small superficial infiltrate, continued to spread, healing in one place, but advancing slowly across the cornea in spite of treatment by caustics, iodin, paracentesis and the thermophore. After three light treatments, it did not stain and showed no further activity during observation.

Mr. F. had been treated elsewhere for four weeks for a corneal ulcer following a superficial injury. A scar covered most of the central cornea, with active ulceration of the dendritic type at its lower border. After four light treatments, combined with iodin applications, the ulcerated area healed and there has been no recurrence.

Richard R., a boy of six, had been treated twice before for dendritic ulcer of the left cornea, once by another oculist and once by me. The last attack of keratitis had been two years before. He showed a large and fairly dense central corneal scar as the result of these attacks. During an attack of whooping cough, he developed numerous points of ulceration near the upper and lower limbus of the same eye. Phototherapy was begun at once, and after four treatments, in addition to three very light applications of trichloracetic acid, the cornea did not stain, the congestion disappeared, and two weeks later the scar of the recent ulceration could barely be made out. The light treatments were given without any trouble to this child, who resisted most violently any local applications, of which he complained bitterly for some time afterwards.

In nearly all these cases, the effect of light treatments on the pain and photophobia was marked from the first. It seems remarkable that a patient suffering from intense photophobia can be made to look fixedly at the uviol light for five minutes with very little discomfort, and that he will afterwards be able to endure ordinary light much better than before.

Another condition which would seem especially adapted to phototherapy is epithelial dystrophy of the cornea, and in the milder forms of this, which show no gross bleb formation, but in which the slitlamp reveals countless minute areas which stain with fluorescein, some good results were obtained. Six such cases were treated, and altho dionin was also given, they seemed to respond more rapidly than those given dionin alone. One case healed in thirteen days after five treatments, and the tension, which had registered 24 Schiötz, decreased to 15. One case which developed during iritis, healed in ten days after seven treatments.

In one case of the severer form, with some deeper corneal opacities, the light treatment was without effect. Two cases of the probably allied condition of recurrent corneal erosion, in whom large blebs were present, were given one to three treatments, but without marked effect, and abrasion and bandage were

resorted to. No other reports have been seen of the treatment of epithelial dystrophy by phototherapy. In the apparently very favorable cases, it is impossible to state that the good results were due to phototherapy, since some cases respond very well to dionin. Here no organisms or virus can be made responsible for the condition, so the apparent effect of phototherapy must be due to a stimulation of the epithelium.

Of types of ulcer other than serpent ulcer and herpes corneae, a good effect was obtained in two cases of corneal scar with degeneration and ulceration over the scarred area, which healed after three and four treatments respectively. In one case of phlyctenular ulcer, the effect was at first good, especially on symptoms, but the condition recurred during treatment. In another, no effect was noted.

Because of the favorable reports of Stock and others on its use in tuberculosis of the anterior segment, phototherapy was tried in three typical cases of sclerosing keratitis probably due to tuberculosis, because of positive tuberculin reactions and negative results of a search for other causes. One very severe case did not return for more than one treatment. The two others, however, showed a quite distinct improvement on phototherapy. In one of these the improvement followed phototherapy alone, tuberculin having been given some time before. In the other case, tuberculin and phototherapy were used together. Both cases were practically quiet when last seen. One case clinically resembling sclerosing keratitis, improved following one treatment, but the condition recurred and was only cleared up permanently after operation on an infected sinus. In these cases, where we do not know whether or not the tubercle bacillus is present in the cornea or sclera, and where a good deal of opaque tissue is opposed to the penetration of light, the assumption of a stimulating effect of the light, producing a dilatation of the capillaries and bringing in a new supply of antibodies, seems the only one which will account for the apparently marked improvement.

In one case of Mooren's ulcer, the progress of the ulcer stopped duing the first three treatments, but then began again in spite of further treatments. No effect was observed in a case of extensive corneal erosion from lime burn and one from acid.

Of the five cases of trachomatous ulceration treated, three responded very favorably to phototherapy. Two of these cases had

resisted all other treatment, and the light appeared to be the factor which turned the scale in favor of healing. In one of these, treatments were given twice daily for three days.

The six cases of blepharitis subjected to light treatment were very severe ones, which had shown no response to yellow oxid of mercury, correction of refraction, and systemic treatment. Four responded very favorably, after 12, 3, 6, and 7 treatments, respectively. In two an autogenous vaccine was given at the same time, but in the other two the light was apparently the only factor responsible for cure. In one of these, the Curay light was used, the quartz applicator being held almost in contact with the lash border and moved back and forth across it for five minutes. In the others, the uviol light was focussed on the same area and moved back and forth for the same length of time. While the results in these cases were apparently very definite, I do not insist upon them, as I have found two to three proper treatments with X-ray to be as effective, if not more so, in obstinate cases, and this treatment is less time consuming for the patient.

In using the uviol light, I have never seen any harmful effect on the lens or deeper structures, nor has the lesion treated ever been made worse by such treatment.

Recently, a series of twenty-eight cases was treated with the Kromayer light, including blepharitis, three; dermatitis, one; hordeolum and Meibomian stye, four; scleritis, three; recurrent erosion, one; smallpox keratitis, two; dendritic ulcer, two; other corneal ulcers, five; chronic conjunctivitis, three; trachoma, four cases. Two sizes of quartz applicators were used in the treatments, one of eight mm. for treating the lids and conjunctiva, and a small one of two and one-half mm. for small corneal lesions. Thru fear of causing painful light reaction, the dosage first used was small, but it was found that forty-five seconds could be safely used on corneal lesions, repeated, if necessary, on other areas of the same cornea, two minutes to the conjunctiva of one lid and fold, and two to three minutes to the whole lash border of each eye. The expected symptoms of light ophthalmia did not materialize. Only a few cases complained of burning after treatments, and in only one case were these symptoms severe enough to cause the treatments to be discontinued. One case developed two punctate corneal infiltrations after his lids were treated, but these healed in twenty-four hours. The

conjunctiva was treated by everting the lid and keeping the cornea under the opposite lid.

The best results were obtained in blepharitis, two cases being practically healed after one treatment, and the other much improved. One case of dermatitis was entirely relieved of itching after one treatment, and the lids became smooth. The reaction around severe deep hordeola seemed to subside rapidly after light treatment and the stye pointed earlier than was expected. One subsided after two treatments without becoming purulent. In one severe dentritic ulcer, healing commenced immediately after light treatments, and a very thin scar resulted. Chronic conjunctivitis with blepharitis responded well when light was applied to both the conjunctiva and lash border. Not enough time has elapsed to speak of definite results in trachoma. Probably the dosage will have to be increased to affect the deeper lesions, and in fresh cases with follicles, the most rapid result will naturally be obtained by expression. In a few cases, where a diffuse thickening remains after expression and other treatment, light applications severe enough to produce a moderate reaction, with swelling of the lids and the formation of a delicate false membrane, have produced a definite decrease in the thickening and a greater smoothness of the lids than had been obtained by scarification or grattage. With a little more courage in dosage, it may be possible to heal some of these old cases in a few treatments, as Lundsgaard and others have reported doing.

While the Birch-Hirschfeld light or Duke-Elder's mercury arc slitlamp are probably the most effective instruments in applying light to corneal ulcers, the Kromayer light or an air cooled light with quartz applicators are almost necessary in the treatment of the conjunctiva and better for treating blepharitis, as much larger amounts of effective ultraviolet may be delivered over larger areas than with the former instruments. I believe the cornea may also be treated effectively and safely with the Kromayer light, by using small applicators and directing the light away from the pupil. I do not believe enough absorption of light occurs in the quartz applicators to necessitate the special applicators used by Duke-Elder, since I have found that enough light is delivered by the Kromayer light with quartz applicator to produce an erosion of the epithelium of the rabbit's cornea after one minute, and a superficial opacity lasting four to six days after two minutes. The Birch-Hirschfeld lamp with uviol filter, on the other hand, after eight minutes produces no erosion.

Summary.

1. Fifty-three cases have been treated by the Birch-Hirschfeld irradiation lamp and twenty-eight with the Kromayer lamp.

2. Beneficial effects of phototherapy, especially with ultraviolet light, may be explained in four ways: by a direct bactericidal effect, by stimulation of the epithelium, by improved nutrition from the hyperemia produced, and by an increase in the antibodies of the blood.

3. General light baths with the mercury vapor arc are of great benefit in phlyctenular keratitis and in ocular conditions due to tuberculosis.

4. Local phototherapy is of benefit in corneal ulcer, tuberculosis of the cornea and conjunctiva, trachoma, and blepharitis.

5. In treating the cornea, the Birch-Hirschfeld carbon arc with uviol filter and quartz lenses is safe, convenient, and produces no unpleasant reactions.

6. Herpetic lesions show the most marked effect of such treatment, some healing after one or two treatments.

7. Epithelial dystrophy is apparently benefited, the early cases observed having all healed under treatment, and more rapidly than those treated by dionin alone.

8. In several cases of sclerosing keratitis, marked improvement has been observed under phototherapy.

9. No marked results were seen in serpent ulcer in this series, but the reports of others would seem to justify a trial of such treatment, especially in early cases, other forms of treatment not being neglected. The length of treatment in these cases might well be increased to ten minntes twice a day.

10. No harmful effects were seen from the Birch-Hirschfeld apparatus in the series.

11. For tuberculosis of the conjunctiva and cornea, use of the Finsen light with special quartz applicators is probably the best form of treatment, but the Kromayer light is also of great value.

12. The Kromayer lamp has proven of value in treating chronic conjunctivitis, blepharitis and deep hordeola, and with a standardization of technic may effect a considerable advance in our treatment of trachoma.

13. In corneal diseases which do not yield to ordinary treatment, such as Mooren's ulcer, phototherapy should be tried.

I wish to take this occasion to thank Dr. F. Lowell Dunn of the Department of Experimental Medicine, University of

Nebraska Medical College, for taking the spectrophotographs and for his valuable advice on technical points.

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DISCUSSION.

Dr. Lawrence Post, St. Louis, Mo.: Dr. Gifford, in his excellent and most interesting paper, has outlined so clearly and comprehensively the known facts regarding the useful ultraviolet rays, with the most probable hypotheses about the action of these rays, that I shall not attempt to go further into that phase of the subject but shall confine myself to a consideration of my own impression of the value of this method of treatment in various types of cases, as derived from an experience of slightly less than two years.

My work has been done with rays from the carbon arc, using composition nickle cored carbons. A uviol glass has been utilized to eliminate possible harmful rays and a quartz lens to condense the ultraviolet rays. I early found that there were obvious disadvantages in the solutions recommended for cooling, so I measured their effectiveness as coolers compared with distilled water, and found that there was an average of only about one degree to the advantage of copper sulphat and ferrous sulphat. So, from that time until about two months, I used distilled water only, for cooling. Dr. Gifford wrote to me about a month ago, that a test in his laboratory showed that distilled water permitted the passage of ultraviolet rays down to 2300 to 2400 angstrom units, and he felt that distilled water in the quartz cell was possibly the ideal cooler.

In the past two months, I have discarded the liquid cooler, so as to make my experience more comparable to his, and I think that without the cooler the results have been more positive.

I find myself in the same situation as Dr. Gifford with regard to making definite statements about results, because the light was always used as an adjuvant to standard treatment and never as the sole method of treatment, so that conclusions can be written as impressions only.

In the first place, I want to congratulate the essayist on his good results as my own have not been so definitely favorable. Not that I have seen many unpleasant outcomes, but rather that there has been a larger percentage of cases in which the rays have seemed to have no effect at all. This may be due to the fact, that most of my treatments during the first year were to cases of iritis and scleritis and episcleritis. My impression was, that I had produced practically no result either good or bad. In the past two months, however, using no cooler and ten-minute daily exposures, a severe scleritis which had resisted all other forms of treatment began to improve at once, and made a recovery in two weeks. The pain disappeared after two treatments. A single case obviously means very little.

I tried the experiment of inoculating the irides of a rabbit with tubercle bacilli and treating one of the eyes, both of which developed typical tubercular nodules, with the ultraviolet rays for ten minutes, twice a day. I could not see that any inhibitory effect was produced clinically on the increasing tubercles in the treated eye, before the death of the animal from generalized infection one month after treatment began. Of course, it was not thought that the treatment to one eye would influence the general tubercular condition, but the experiment was made to see whether the rays would inhibit the growth of the tubercles in the exposed eye. Continued experience has led me to the conclusion, that treatment to the eye alone is insufficient in deep lesions, and that general light therapy must be given at the same time in order to produce a good result. My experience in these cases corresponds with Dr. Gifford's lack of success.

Corneal lesions have undoubtedly shown the greatest benefit from the light, especially the superficial, acute lesions. Of these, the herpetic type especially has been responsive.

A number of trachomatous corneas were treated, with a slight benefit to some. There was usually a considerable reaction following the first few treatments, in one case seeming to be the cause of a drop in vision from 20/120 to counting fingers at two feet. This was followed by a slow clearing after discontinuance of the rays. The course of these cases is so notoriously full of ups and downs, that the exact relationship to the light treatment is always uncertain. Application to the follicles has seemed to produce little benefit in my hands.

Recent interstitial corneal lesions have occasionally been apparently much helped, the improvement following a period of reaction with definite increase of the clouding. On the other hand, many such cases have shown no effect whatever, and I have not yet come to any conclusion as to which cases will react and which will not.

Corneal scars have been treated by some with apparent success. One St. Louis ophthalmologist has treated two patients in whom there was a

marked visual improvement, which he thinks was due to the light. My own results with old corneal scars has been nil, tho I have been impressed, in the course of careful observation of these cases from week to week, at the variability of vision, this ranging in a typical case all the way from 20/60 to 20/120 and back again.

One type of trouble that has shown very definite benefit in all of the cases in which I have tried the light, has been vernal conjunctivitis. Both subjectively and objectively, the trouble has been greatly improved. A few treatments in the milder conditions have almost completely relieved the distressing itching. I have not used it in severe cases, such as where the cobblestone appearance of the upper palpebral conjunctiva has been present.

In conclusion, I feel that we have a real therapeutic agent in ultraviolet light, but one that must be studied a great deal longer before we shall know very much about it. In this therapy, as in any other, it is so easy to be misled by the wish being father to the thought.

Dr. C. W. Rutherford, Iowa City, Ia.: My experience with light therapy in diseases of the eye is less than that of Dr. Gifford. He omitted nothing of importance from his paper. I am acquainted with the literature of the subject, and I wish to emphasize the economic side of the question.

Several styles of generators, with their individual equipment, are required to establish an outfit that can be considered adequate to meet all situations, and the cost of installing a representative group of instruments is as yet out of proportion to demonstrable superiority over commoner methods of treating ophthalmic diseases in private practice. Few private offices can afford space for such a multiplicity of instruments and the time to operate them properly.

A few years ago, Dr. Jackson editorially curtailed the probable usefulness of the slit lamp as then constructed, while Dr. Wuerdemann assigned it to an honored place in the attic, with other ophthalmologic relics. They must have been in error, for we do not find used slit lamps advertised for sale, like ophthalmometers and perimeters. Already, we frequently see offers to dispose of used and "never used" light therapy equipment.

The new in therapeutics has always lured the enthusiast who must at any cost maintain a position in the front rank of medical progress, as well as those whose custom it is to commercialize each innovation in practice; neither is mentally or ethically equipped to render the most intelligent service. A tragic example is the present prevalence of optic atrophy in patients treated exclusively with salvarsan some twenty years ago. Roentgen rays and radium have been employed therapeutically for several years, and we still see freshly wrought examples of their misuse. Phototherapy is comparably less dangerous, perhaps, and it will develop into a trusted agent, unless discredited and condemned by unforunate effects in the hands of those who employ it without first becoming qualified to use it.

Examination of the literature suggests that the ophthalmic diseases for which light therapy is used are prone to relapses when treated by any means. We must learn to select with some certainty the kind of generator, screens and other essentials, and the dosage to be applied, with

confidence, to suit the individual patient as well as the character of the disease. Few private practitioners are so fortunately situated that they can profitably equip themselves for adequate study. Even institutions that are comfortably supported and well supplied with assistants might find it burdensome to obtain and supply room for all of the equipment now on the market, especially when we consider the space required by desirable and important aids to diagnosis.

Light therapy will occupy a prominent place in ophthalmic practice when the several wave frequencies shall have been assigned to special tissues and conditions and the apparatuses combined or simplified, when the response of the patient can be anticipated with reasonable assurance, and after much patient institutional research work shall have been done by Dr. Gifford and others equally zealous in their quest for fundamental facts.

Dr. William H. Crisp, Denver, Colo.: The last few years have seen a number of additions to our armamentarium for treating some of the more obstinate inflammatory conditions of the anterior segment of the eye. Of these new resources, in the long run, the tendency will probably be to resort to those which are at the same time effective, simple, and relatively inexpensive.

If we were to expand the title of this present subject to radiotherapy rather than light therapy, we could include heat rays, which in the form of treatment outlined by Dr. Gifford apparently have been found to add to the efficiency of the ultraviolet rays. We could also include the use of the X-ray.

In the instructional courses, we have listened to Dr. Shahan's statement of his use of the thermophore. It seems to me, the reports of results obtained with the thermophore in sluggish corneal ulcer and in a number of other conditions of the anterior segment are comparable with those obtained with ultraviolet rays in similar lesions. There is no question that the thermophore or some similar device is much simpler for the average ophthalmologist to use than the ultraviolet ray.

On the other hand, I think we ignore too much the possibilities of fractional doses of X-ray applied to some of these conditions in the eye. I have had a limited experience in this direction, because I know that most of these conditions will yield to methods that have been in use for many years, and therefore I resort only exceptionally to special methods. But in a limited series of sluggish corneal ulcers, and in one or two other conditions in the anterior segment, I have found that a one-third erythema dose of X-ray applied by a thoroly reliable roentgenologist proved very efficacious. Recently, I had a patient with an episcleritis which was not extremely severe but was persistent. It did not yield to the use of homatropin every hour for a day or so, but the patient got well practically within twenty-four hours without homatropin under the use of small doses of X-ray.

Dr. S. Judd Beach, Portland, Me.: My experience with ultraviolet light in that type of chronic recurrent keratitis resulting from tuberculosis which slowly goes on to complete destruction of vision makes me feel that we are depriving these patients of their best remedy if we do not attempt to familiarize ourselves with this remarkable new therapy. As you have perhaps noticed, many of the reports today have been made

with the statement, that it is a little difficult to evaluate the use of the light on account of the fact that other regular methods of treatment were being used at the same time. Altho my cases are not numerous, the fact that I was using no other treatment than the light at the time when they recovered, makes me feel more strongly that it is a valuable remedy. I am grateful to Dr. Gifford for giving us more details about its use.

Dr. S. B. Muncaster, Washington, D. C.: In the use of the infrared rays, one factor that is often overlooked is the temperature reaction. Three years ago I had a severe case of gonorrheal ophthalmia in Georgetown University Hospital that did not respond well to treatment. Accordingly, I tried the infrared rays. The eyes were protected with six layers of gauze, and the lamp was held twenty inches from the eyes for twenty minutes. Within six hours, the temperature was reduced one and one-half degrees below normal, and it did not return to normal until the third day. I then repeated the treatment, and the same result followed. During the period while the temperature was down, the child was in a poor condition physically. Altho the effect upon the infection was beneficial, I stopped this treatment because of the constitutional disturbance caused by the light.

Any of the lights similar to the infrared lamp should be used with caution, because some disturbance unwished for might be obtained. For example, an individual with low blood pressure needs all the vitality he has, and a drop in temperature attended by constitutional disturbance caused by the application of the lights would not be very desirable.

Dr. Sanford R. Gifford, Omaha, Neb. (closing): I just want to say that this is certainly not the treatment for most cases, but it is of value in certain selected cases. The cases we would like to see it used on are not those that yield easily to other therapy, but those which usually heal with a certain amount of scar tissue. The cases I am most interested in are those of herpetic keratitis. These have healed with a very thin scar, and I have found other methods of treatment often leave a heavy scar. Relative to these cases, I speak of the Birch-Hirschfeld apparatus, which I believe is the best for treating the cornea.

I am glad Dr. Rutherford brought out the question of economy, because most of these elaborate instruments can be used only in clinics. But the Kromayer lamp is something which every dermatologist has. It is almost necessary if you are going to treat cases of severe blepharitis with phototherapy.

I am sure you will be gratified if you will refer some of these obstinate cases to a skin man for several three-minute treatments of the lids with the Kromayer lamp, as I know some of them will clear up which had resisted ordinary treatment.

I am glad Dr. Beach spoke with encouragement, because I think you will find the treatment of value in a small series of cases, altho not in cases of serpent ulcer.

THE FREQUENCY IN THE NEWBORN OF INTRAOCULAR HEMORRHAGES AND THEIR RELATIONSHIP TO INTRACRANIAL TRAUMA.*

MAX W. JACOBS, M.D.

ST. LOUIS, MISSOURI.

That intraocular hemorrhage, as well as intracranial trauma and bleeding, is a very frequent occurrence in the newborn has been shown by numerous investigators. In a paper on "Retinal Hemorrhages in the New Born" read at the American Medical Association's meeting in 1924, I reported that twelve per cent of the infants examined at birth showed retinal hemorrhages. While some writers believe that a particular type of presentation may have a definite relationship to this phenomenon, my data gave no support to such a viewpoint. A recent study of the eyes of these children, now ranging in age from three and a half to five and a half years, showed no impairment of vision as the result of the retinal hemorrhages found at birth. Intracranial injury at birth, with or without hemorrhages, on the other hand, is a much more serious matter. It is, evidently, one of the most important factors in the mortality of the newborn. Cruveilhier, early in the eighteenth century, claimed that cerebral hemorrhages occurred in one-third of all still born infants. Capper concludes his review of the literature and his study of cerebral hemorrhage in the newborn with the following statement.

"The clinical and pathologic data show the general agreement concerning the enormous frequency of cerebral injury at birth. Wohlwill found that cerebral trauma at birth causes a loss of human life so great, that it may be considered as one of the most widely spread diseases. Next to tuberculosis, disease of the nervous system due to cerebral trauma at birth is the most widely prevalent disease from the point of view of frequency. Its mortality is one of the greatest. Schwartz said that ten per cent of all the newborn infants who at the beginning of the delivery were viable per se, died at the end of the first month as a

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result of cerebral trauma at birth. Pathologists are of the opinion that this great loss of human life is overwhelmingly caused by the traumatic injury of the brain at birth; all other causes such as congenital syphilis, asphyxia due to cord around the neck, asphyxia thru anomalies of the delivery, primary infections and malformations, are present in a small number of cases when compared with the number in which trauma is the cause. Immature and premature births, of course, form the most important contingency in this group."

Other writers who have called attention to the frequency of intracranial hemorrhages at necropsy are Van Cleave, Jaschke, and Heidler. Van Cleave, averaging the reports of nine investigators, found a frequency of 22.3%. Jaschke gave 1% as the number of children who died at birth from head injuries. If the premature be subtracted, the final number is reduced to .22%. In his own series, however, Heidler found as large a percentage as .88%. Capper, in discussing microscopic and macroscopic postmortem reports, assumes, as a rule, that only the macroscopic hemorrhages are the cause of some still births, neonatal deaths, or deaths within the first month of life. He also points out the close relationship between the degree of the development of the immature infant and the frequency of the occurrence of cerebral hemorrhage.

With the thought that some relationship might be traced between intracranial trauma and hemorrhage and intraocular bleeding, a series of premature and still born infants were examined at Washington University. Postmortems were done in each case and the heads opened by the Beneke method, so that intracranial lesions could be more easily observed. Paraffin sections were made of the eyes, and the accompanying table shows the result of these studies.

Of fourteen children examined, all showed intraocular engorgement or hemorrhage. In the choroid of each, were vascular phenomena varying from engorgement to hemorrhage. Twelve pairs of eyes had retinal hemorrhages. One of the two having no hemorrhage lived four days. It may have had a retinal hemorrhage when born, as these lesions sometimes disappear a few days after birth. The other pair of eyes free from hemorrhages were those of a macerated fetus. One would not be likely to find here the changes due to the trauma of birth, as they are found in a living child or one which died during labor.

Sex of Child	Life of Child	Weight at Birth	Length at Birth	Evidence of Lues in Child	Intracranial Findings	Eye Findings
M.	¾ hrs.	3740 gm.	54 cm.	None	Intracranial hemorrhage. Laceration of falx cerebri. Laceration of left tentorium cerebelli. Questionable whether amount of blood present could account for death.	Considerable engorgment in choroid. A few engorged retinal vessels and hemorrhages.
F.	Still born	2720 gm.	46 cm.	None	Tear of falx and tentorium. No intracranial hemorrhages.	One eye showed some choroidal engorgement. A few engorged retinal vessels and hemorrhages.
F.	Still born	2270 gm.	46 cm.	None	Tear near center of falx. Laceration of tentorium on left side. Blood in cerebellar fossa.	Marked choroidal engorgment. Engorged retinal vessels. Some retinal hemorrhages.
F.	Four days	2110 gm.	45 cm.	None	Spina bifida and meningocele. Edema of meninges.	Choroidal engorgement. No retinal hemorrhages.
M.	Still born	2880 gm.	51 cm.	None	Intracranial findings negative.	Moderate degree of engorgement of choroid. Occasional engorged retinal vessels. Few retinal hemorrhages.
M.	Still born	3300 gm.	51 cm.	None	Laceration of tentorium cerebelli.	Choroidal engorgement. Retinal hemorrhages.
F.	7 hrs.	2590 gm.	50 cm.	None	Laceration of falx cerebri. Free blood upper surface of tentorium.	Large choroidal hemorrhage. Extensive retinal hemorrhages.
F.	Still born	4680 gm.	55.5 cm.	None	Spina bifida with meningocele.	Choroidal engorgment. Some diffuse retinal oozing. Occasional retinal hemorrhages.
M.	Still born	3420 gm.	55 cm.	None	Fracture of 5th cervical vertebra.	Marked choroidal engorgment. Occasional retinal hemorrhages.
M.	Still born	4550 gm.	54 cm.	None	Intracranial findings negative.	Choroidal engorgment. Few retinal hemorrhages.
M.	Two days	3460 gm.	52 cm.	None	Intracranial hemorrhage. Laceration of tentorium cerebelli, right and left.	Marked choroidal engorgment. Large retinal hemorrhages.
M.	Macerated fetus	1950 gm.		Bone section not satisfactory. No spirochete in tissue	Tentorium on left side showed congenital defect and not a laceration of falx in central portion.	Marked engorgment of choroid. No retinal hemorrhages.
M.	Still born	2600 gm.		None	Intracranial findings negative.	Moderate choroidal engorgment. An occasional engorged retinal vessel. A few small retinal hemorrhages.
F.	2½ hrs.	1830 gm.	43 cm.	None	Intracranial findings negative.	One eye showed moderate infection of choroid and no retinal hemorrhages worth noting. Other eye showed some retinal hemorrhages.

No.	Color	Age	Parity	Gestation	Pregnancy	Kind of Labor	Position	Length of Labor	Wassermann
2597	B.	17	P.	41 weeks	Normal	Spont.	L. S. A.	16 hrs.	Negative
2699	B.	32	M.	40 weeks	Normal	Spont.	R. L. O.	10⅙ hrs.	Negative
2710	B.	15	P.	34 weeks	Normal	Spont.	L. S. A.	12⅚ hrs.	Negative
2711	B.	30	M.	40 weeks	Normal	Spont.	L. S. A.	Out-patient	Not taken
2728	B.	30	M.	40 weeks	Normal	Spont.	L. O. A.	10⅓ hrs.	Negative
2729	W.	30	M.	40 weeks	Normal	Spont. Forceps Mid. Double Application	R. O. P.	6 hrs.	Negative
2779	W.	26	P.	43 weeks	Hypertension Albuminuria	Difficult. High Forceps	R. O. P.	16 hrs.	Not taken
2812	W.	23	M.	40 weeks	Normal	Induced	R. S. A.	11½ hrs.	Negative
2832	B.	38	M.	40 weeks	Normal	Spont.	Podalic Version	11¾ hrs. Prolapsed cord	Not taken
2864	W.	18	P.	40 weeks	Normal	Spont.	R. M. P.	10 hrs.	Negative
2874	W.	23	P.	41 weeks	Normal	Induced Low Forceps	F. O. P.	25⅙ hrs.	Not taken
3144	B.	23	M.	40 weeks	Normal	Died Next Day	Breech Potter Tech.	140 hrs.	Negative
3153	W.	33	M.	34 weeks	Normal	Spont.	S. L. A.	2 hrs.	Negative
3180	W.	28	P.	27 weeks	Normal	Spont.	L. O. A.	5⅓ hrs.	Not taken

Four children were premature or immature, but the intracranial and intraocular findings showed no variation from those found in children born at term. Wassermanns done on eight of the fourteen mothers were negative, and examination of the tissues of all the children failed to show evidence of lues. Color, parity, age of mother, and length of labor seemed negligible factors. In the cases in which forceps were used, tears of the falx or tentorium were found, and in two of these, the choroidal and retinal lesions were especially pronounced. Twelve of the fourteen were cases of spontaneous labor. The only case in which pregnancy was complicated (hypertension and albuminuria) was one that required high forceps. This infant showed an intracranial injury as well as large choroidal and retinal hemorrhages.

Six of the presentations were breech. One of these was a spina bifida with meningocele, and showed choroidal engorgment and retinal hemorrhages. A second breech, a premature baby, with no other apparent complicating factor had a tear of the falx, laceration of the tentorium and blood in the cerebellar fossae. The choroid and retina were engorged and there were retinal hemorrhages. The third of these presentations was a spina bifida with meningocele, the eyes having choroidal engorgement, but no retinal hemorrhages. This baby lived four days. The fourth, born of a past term primipara, showed intracranial hemorrhage, laceration of the falx and tentorium with considerable engorgement of the choroid and a few retinal hemorrhages. Number five of this group was a macerated fetus with a laceration of the falx, marked engorgment, but no hemorrhages in the retina. The sixth, a premature still born, had no intracranial pathology, but there was some engorgment of the choroid and a few retinal hemorrhages. In short, the breech cases, as such, showed no greater amount of intraocular hemorrhages than did the other types of presentation. The intracranial findings of the whole series showed tears of the falx in six, tears of the tentorium in five, and macroscopic intracranial hemorrhages in three.

Vasolability, according to Capper, is one of the chief predisposing causes of intracranial and other forms of hemorrhage in immature infants. He says, "Lack of elastic tissue in the blood vessel walls may be the reason that the blood vessels of the immature as well as the premature infant are easily traumatized. Incomplete architectural development of the capillary system may

be a predisposing case of hemorrhage in immature infants. Archicapillaries are a fetal stage of the mature capillaries, and in most normal infants, the archicapillaries are not found at birth. Doxiades found a persistence of the archicapillaries in most immature infants. It is therefore possible that this antenatal state of the capillary bed is not of sufficient strength to withstand the trauma associated with uterine contractions and passage thru the birth canal." Sicherer suggested that obstruction of circulation takes place in a definite area with the result that retinal hemorrhages occur on the corresponding side. My investigations published in a previous paper failed to substantiate this idea.

In considering the data gathered in this paper, the striking fact is, that I found such a large percentage of retinal hemorrhages. The idea that hemorrhages in the premature may be due to some vascular weakness, as was suggested above, is not borne out in this piece of work. A study of all the factors involved in this series would seem to show no single one of them responsible for simultaneous intraocular and intracranial hemorrhages.

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DISCUSSION.

Dr. William Zentmayer, Philadelphia, Pa.: Dr. Jacobs is to be congratulated upon having supplemented his interesting paper of 1924 by this valuable study undertaken to determine a possible relationship between intracranial trauma and hemorrhages and intraocular bleeding. A summary of his findings shows that:

There was no case in which intracranial hemorrhage was present without associated intraocular hemorrhage, but there were eight cases of intraocular hemorrhage without associated intracranial hemorrhage. There were four cases of associated intracranial hemorrhage and intraocular hemorrhage.

In only two cases were there neither intracranial nor intraocular hemorrhage, and in one of these the infant was macerated, and in the other hemorrhage might have been present and disappeared.

Of this series of fourteen premature and stillborn infants, at least 86 per cent showed intraocular hemorrhage. In 28 per cent there was associated intracranial and intraocular hemorrhage, and in 57 per cent there was intraocular hemorrhage without associated intracranial hemorrhage.

The author found in his former series 12 per cent of newborn infants to have retinal hemorrhage, whereas in this series the percentage is at least 86. This difference may be explained by the fact, that in the former the findings were ophthalmoscopic, while in the latter they were microscopic. The much higher percentage of intraocular hemorrhage than intracranial may be explained by the better support afforded the intracranial vessels by the firmer tissues of the brain, and by intracranial tension.

It is interesting in this connection to consider the incidence of hemorrhage in mature and premature infants. Cruickshank gives the statistics in 200 mature and 200 premature: 80 per cent of the mature and 66 per cent of the immature had hemorrhages into tissues of some kind. In about 60 per cent of all these, the hemorrhages were gross; in 32 per cent they were intracranial; in 30 per cent there were tears of the tentorium of the cerebrum or cerebellum. In Dr. Jacobs' series, the percentage of tears of the tentorium was 50.

In a symposium on birth injuries of the eye of the newborn before the Ophthalmological Society of the United Kingdom in 1826, Juler, in discussing retinal hemorrhages, drew the following conclusions:

1. Retinal hemorrhages occur during birth in from 10 to 20 per cent of children.

2. The main factor would seem to be a congestion of the retinal veins, induced thru the cavernous sinus, which is subjected to increased intracranial pressure during the birth process.

3. The incidence may be determined by the number and width of the lateral anastomoses of the central retinal vein in its course thru the orbit to the cavernous sinus.

4. Abnormalities of labor which tend to increase the cranial pressure appear to exercise little effect on the incidence of retinal hemorrhage

It has long been thought by some, that in certain instances, congenital amblyopia may be due to natal hemorrhages occuring at the macula. In this connection, it would be of value to know in what percentage of the cases examined microscopically by Dr. Jacobs hemorrhages were found in this area.

When we consider the statistics of Cruickshank as to the frequency of hemorrhage into the tissues in general, in mature as well as premature infants, in connection with those of Dr. Jacobs in the eyes of premature infants, it would seem to indicate that these hemorrhages are a normal result of labor, and there is no need to look for a contributing cause in the nature of the labor, or to any pathologic condition of the blood vessels, to explain them. There may be, however, an anatomic reason in the structure of the vessels at this period of life.

Dr. Arthur J. Bedell, Albany, N. Y.: When Dr. Jacobs read his first paper, I prepared a discussion by examining the eyes of a large number of newborn babies in the Brady Maternity Hospital. This report is included in his original communication.

For three reasons it is a pleasure to speak at this time: First, because Dr. Jacobs has been able to see and record the late appearance of some of his cases, and to establish the fact that the intraocular hemorrhage of the newborn is not a cause of impaired vision. Second, because he has continued his work, amplified it, and finally become interested in the immense group of hemorrhages resulting from birth injury. Third, because it was my privilege to see in the Scientific Exhibit of the American Medical Association at Minneapolis, this Summer, the collection of fetal heads presented by Dr. W. A. O'Brien of the University of Minnesota Medical School. Thru the kindness of Dr. O'Brien, four of these heads have been placed on a table in the back of this room. I am able to express his opinion and also that of his associate, Dr. F. L. Adair, "That the injury to the skull results during any type of labor in which distortion occurs. Hemorrhages and lacerations of the falx and tentorium result, and intracranial injury with hemorrhage follows." I am glad to acknowledge my indebtedness to Dr. O'Brien.

The work of Dr. Jacobs in this and allied ophthalmoscopic interrelations with abstetric problems will be more appreciated as other observers see the value of eyeground studies. I am convinced that good will come from such careful clinical compilations as the author has presented.

Dr. F. Park Lewis, Buffalo, N. Y.: The number of children suffering from head injuries would seem to justify the statement made by the late Dr. McMurtry, of Louisville, that child birth is a surgical procedure. We frequently find in adults results of macular hemorrhages causing imperfect sight, which are produced by pressure on the soft bones of the infant's head, just as neurologists trace profound nervous disturbances to similar birth traumatisms.

I rise merely to suggest that obstetric practice has been developed in the last few years in a way that may have an important bearing on this subject. Ten years ago, I heard a discussion of Caesarian section in which five cases were reported for a year, and the surgeon was criticized for doing so many. Last year, in one hospital in Buffalo, in 1,000 obstetric cases, 150 were delivered by laparotomy. The results are so satisfactory in such a large number of cases, the child simply being lifted out of the uterus without the slightest disturbance of the head and with none of the deformities, none of the hematomas which are found in normal deliveries, that I am led to suggest that it might be worth while for such a society as this to have a general study made by ophthalmologists, neurologists and obstetricians, to determine whether Caesarian section should not be more often performed than it is, and whether it will not prevent such injuries as those which we find in the child delivered in the normal way.

Dr. Max W. Jacobs, St. Louis, Mo. (closing): Hemorrhages into the retina in this series of experiments were certainly not limited to any particular region. They were scattered indiscriminately thruout the retina. The terrific engorgement found in the choroid in every case was a generalized thing. I want to stress the fact that hemorrhages certainly could not be localized in the region of the macula.

Some eighteen months ago, I was able to get in touch with thirteen of the twenty-three children who at birth had shown retinal hemorrhage, as reported in my previous paper. These children, when last seen, ranged in age from three and one-half to five and one-half years. I was able

in every case but one, a Mongolian idiot, to do fairly accurate refractions and make fairly dependable tests of vision. These children were seen repeatedly. With the exception of one case, where there was a certain amount of amblyopia, I can state definitely, that there was every reason to believe that hemorrhages had had no influence upon visual acuity. The one case that had a considerable degree of amblyopia had had a hemorrhage fairly close to the region of the macula, a larger hemorrhage than in any of the other cases. Many of the others had had small hemorrhages in the region of the macula, without any influence on visual acuity.

Some years ago, I saw a patient with tremendous hemorrhages into the retina. They were so large that it was difficult at times to see details of the eyegrounds. One eye was apparently sightless, the other had some vision. This child has been in the hands of another St. Louis oculist, and I understand has some vision in one eye. This child had a coloboma of both optic nerveheads, and it was supposed at the time that the terrific amount of hemorrhage probably was due to abnormalities in the formation of vessels leading to the optic nerve. With the exception of this case, certainly none of my own series would seem to give us much cause for worry as regards eventual acuity of vision. Only very large retinal hemorrhages at birth, and especially those in the region of the macula, would seem of importance.

TRAUMATIC PROLIFERATING CHORIORETINITIS.

ELMER P. WEIH, M.D.

CLINTON, IOWA.

Since traumatic proliferating chorioretinitis is seen so rarely in peace times, and since the subject is so interesting, I think it warrants presenting.

Our patient, a boy of fifteen years, came to me on February 7, 1927, twenty-seven days after having been accidentally shot with a 22 rifle. There was a small scar 1/16 of an inch in diameter, the point of entrance located in right temple, on a horizontal line exactly one inch from outer canthus of right eye.

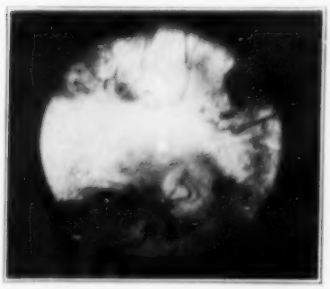

Fig. 1 Right Eye. Periphery.

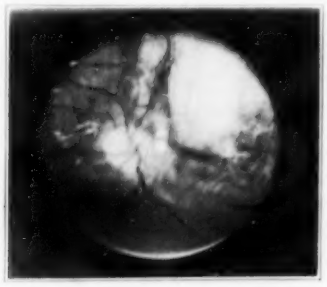

Fig. 2 Left Eye.

There was no point of exit, however, point of bullet could be felt just thru skull in left temple, 1½ of an inch posteriorly, and ⅜ of an inch above external canthus of left eye. Lids of both eyes were swollen. Conjunctiva of left eye markedly edematous, projecting out between eyelids. Conjunctiva of right eye red and swollen. Numerous adhesive bands obliterating lower cul-de-sac and attached to conjunctiva of eyeball, both on outer and nasal side of eyeball. There was some discoloration of skin of upper and lower right eyelids, yellowish blue tints, probably due to blood pigment absorbing. Large subconjunctival hemorrhage upper and outer quadrant of right eye. Pupil of right eye seven mm. in diameter. Left eye eight mm. in diameter. No reaction

to light. Eyes do not move in any direction. Cornea, anterior chamber, and lens normal. Virteous fairly clear. Optic nerve-head white. Retina almost completely covered with blood. Macula region contains blood. Numerous black pigment spots scattered here and there, with an occasional white necrotic spot of retina showing thru. Much pigment scattered thruout the

Fig. 3 Right Eye.

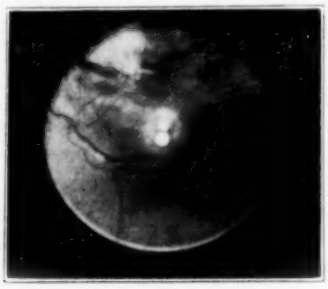

Fig. 4 Left Eye.

fundus. No light perception in eye. Left eye shows cornea, anterior chamber and lens normal. Vitreous is clear. There is much fresh and old blood in upper part of fundus. Light localization fairly good. Makes a few mistakes. Counts fingers in upper field of vision. Roentgen ray examination shows a tract of fine lead particles across both orbits apparently cutting right

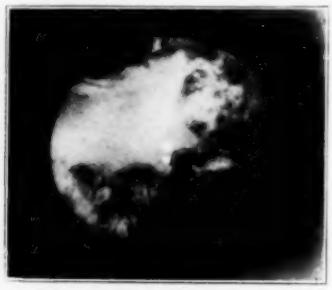

Fig. 5 Right Eye. Periphery.

optic nerve, perforating ethmoidal cells and passing just above left optic nerve region. Here the bullet broke into two large pieces, one going upward and back, the other downward and forward. Blood Wassermann examination was negative.

April 10, 1927. Patient can open right eye 8 mm., left eye 5 mm. Right eye: Blood in vitreous is all absorbed. Blood

vessels where they come into disc are distorted toward the nose and downward. Vessels supplying the upper and outer part of retina have been torn away. There is a large tearing of choroid extending upward and inward, half the distance to ora serrata. There is much pigmentation. Much of the outer and upper quadrant of fundus is a white membrane with irregular blotches of pigment in it. Blood vessels can be traced only a short dis-

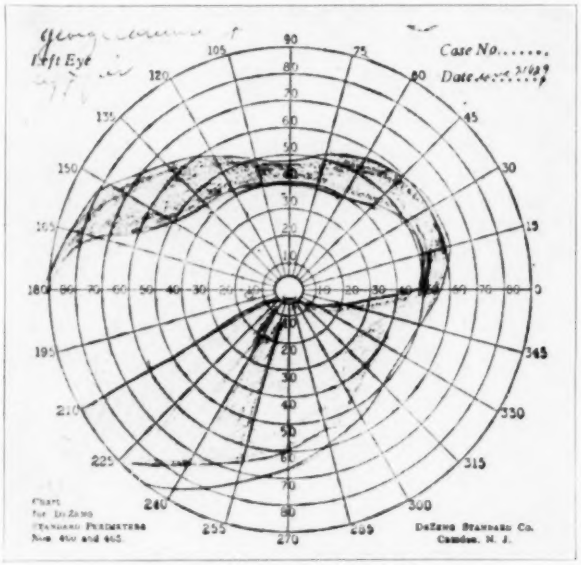

Fig. 6.

tance, when they lose themselves in this torn area. Fundus of left eye shows a large irregular blotch of black pigment in the upper and outer part with this white membrane showing thru here and there. Blood is practically all absorbed.

On May 31, 1927, boy can open right eye 10 mm., left eye 7½ mm. Can move both eyes in all directions. Right eye is blind. Left vision equals 20/200. Lantern slides will show condition of fundi and field of vision.

Collins and Mayou state that a bullet in a gunshot injury may enter the orbit on temporal side, pass thru it, across the ethmoidal sinuses, thru the orbit on the other side, and emerge from its outer wall without in anyway jeopardizing the life of patient.

Dimmer states that direct choroidal rupture and socalled chorioretinitis proliferans traumatica or retinitis scolopetaria pre-

sent similar changes. Direct choroidal rupture occurs at the point of impact, differs from indirect rupture in having no deep fissures, and is rather superficial and often of considerable extent. While indirect rupture only occasionally tears the retina, this is common in direct rupture. As a result, we find more copious hemorrhage about the rupture, not only in the choroid and between this and the retina, but also in the vitreous.

McKee states that this proliferating chorioretinitis does not follow hemorrhages of the vitreous such as recurring hemorrhages in young subjects, but follows hemorrhages of the choroid and retina produced by rupture.

Fig. 7.

Chou says, that in gunshot injury of the orbit, the retinal tear lies parallel to the direction of the force. The mechanism is similar to that of the injury coming from the front.

Wurdemann states these cases are associated with a direct traumatic origin, together with a rapid coagulation of blood and very slow absorption.

De Schweinitz says that lacerations of the choroid and retina, or of their vessels, or of both followed by hemorrhage beneath the retina and into vitreous are common. They depend in part on the stretching of their membranes, and on the vibrations in the vitreous. Gunshot wounds of the orbit cause extensive

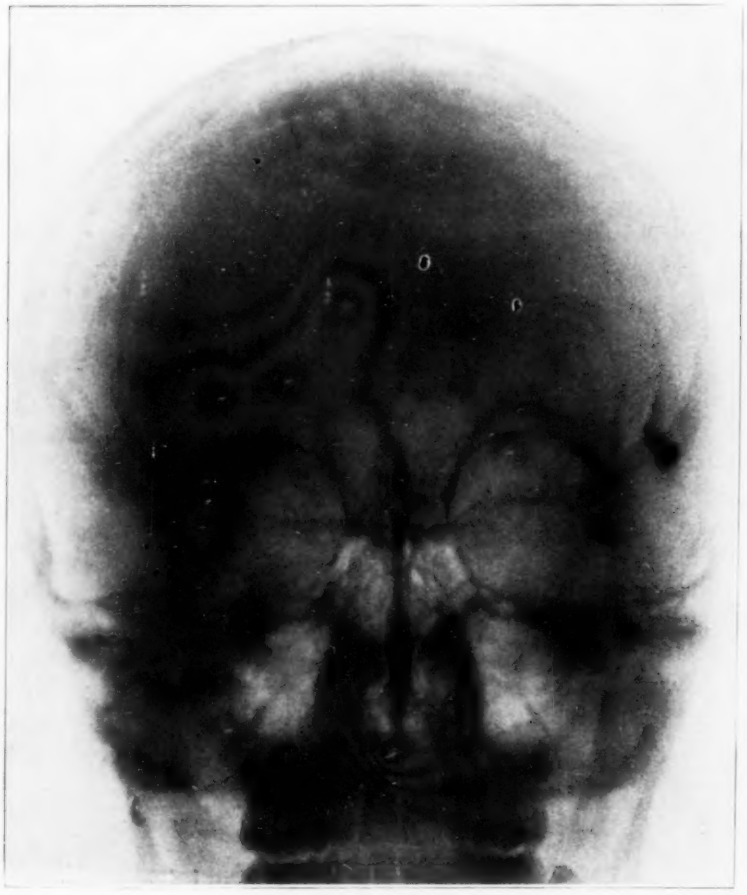

Fig. 8.

changes and lead to chorioretinitis proliferans. Direct choroidal and retinal ruptures caused by the impact of large foreign bodies appear as larger irregular bright spots, often triangular in shape, with point directed towards the disc. Gunshot wounds of the eye present wholly different and much more extensive changes.

Lister states these glistening white spots are particles of coagulation necrosis brought about by the rupture of retinal and choroidal vessels, these cutting off the blood supply and nutrition to certain areas. In peace times, these ruptures are seen in cases of suicidal attempts causing transverse orbital shot wounds, but has also been observed in orbital wounds from the front.

New wounds of this kind are often difficult to study because of the profuse hemorrhage in the vitreous and the preretinal area. Even where minor damage results, the vision is much impaired following a blow on or near the eye; this impaired vision disappears after a while. The perceptible changes consist of hemorrhages upon and beneath the retina, upon and beneath the choroid, extensive clouding of the retina, of indications that point to the rupture of the choroid and the pigmentation of the retina and the choroid. Later there appears the scar tissue in the retina and choroid and the resulting conditions from its contraction, the consequent changes in the blood vessels, and distortion of tissues.

At the point of injury, which is usually near the nervehead and vicinity of macula—it may be anywhere—one can often note very extensive white discolored spots, upon and near which hemorrhages are observable in new wounds, and sometimes months later. These white spots show extremely irregular, yet rather definite, tho not clear cut edges, with most diverse shapes. From them, rather long processes often reach out in various directions, which if numerous, give the edge of the white section the appearance of contiguous arches or arcades. The retinal vessels extend over all or most of the white spot or reach into it at various places and lose themselves in it. The curving of the retinal vessels at the edge of the white mass, the parallactic shifting and the refractive difference, show that at this place the membranes of the eye have grown thicker, for the refraction difference sometimes indicates a prominence of one or two mm. Even in older cases, considerable hemorrhages are occasionally observable, which are in part to be considered preretinal, if they lie clearly outlined in front of the white mass, as is indicated by

parallactic shifting with relation to the white mass. Such hemorrhages may project vesicularly into the vitreous. Other hemorrhages lie in the white spots themselves and are not always clearly outlined. Of course, retinal, as well as preretinal hemorrhages, occur also entirely outside the areas affected by the white spots. Within the white spots, and more often at their edge, irregular masses of pigmentation are observable, especially in older cases.

Round these white masses and projecting over them, one sees very irregularly shaped spots, which appear light tinged with yellow. Within them often lie single base choroidal vessels and irregular pigmentations. Not rarely these light colored sections, which are to be considered as representing choroidal tissue ruptures, have pigmented edges. Proceeding from the center of the area affected, they sometimes surround the nervehead in the shape of an irregular ring. In cases where they do not show, they are probably obscured by the white masses.

The general picture presented above often shows many irregularities in individual cases. The large white masses are often interrupted by round or oval light red spots, clearly outlined, the size of which often attains ½ papilla diameter. Their clear outline and their regular form show them to be no ordinary hemorrhage; they appear rather to be holes in the white mass, thru which the red fundus becomes visible. They may, however, be blood filled cavities in the exuding matter.

Because the retina and choroid are ruptured, the retinal vessels at certain places show interruptions, also dilatations and contractions, the arteries appearing decidedly contracted or entirely obliterated, changed into white stripes, while the veins running in the same direction are often dilated. There may appear vessels which by their origin and course are shown to be newly formed. Among these are vessels which arise within the scarified areas clearly without any connection with the central vessels of the retina, and which therefore probably arise from the choroidal vessels implicated in the scar. That, under such conditions, the walls of the vessels undergo great changes is selfevident. The vessels are often changed into white stripes or strings. Abnormalities in the course of the retinal vessels result from the fact that the shrinking of the scar draws the retina over the nervehead, so that the vessels of diametrically opposite portions of the retina are drawn over the nervehead and form loops on it. The drawing-over of the retina in such a manner that it forms

a fold upon the papilla has been anatomically demonstrated by v. Szily.

As a result of this shrinking process, one can occasionally observe retinal folds leading out from these places into the unaffected retinal area. They appear as gray turbid stripes radiating into the surrounding retina and are also evidenced by the retinal reflexes. This is most clearly observed with the red free light. Partial loosening of the retina may be observed in areas surrounding retinal-choroid ruptures. Folds may also occur in the choroid.

With all these changes, the disc is pale, sometimes only in that portion of the disc that faces the retinal rupture. In cases where the disc is surrounded by injured tissue, it is generally pale or even white. The course of the vessels sometimes indicates that the papillary tissue is torn.

The changes that appear later are: first, the clearing of the vitreous from which the cloudiness caused by the hemorrhages gradually disappears, second in the shrinking and diminution of the scar like retinochoroidal changes, and third the appearances of heavier pigmentation in the scarred areas.

Lagrange says that classical proliferating retinitis is produced by organization of the extravasated blood, by the appearance of membranes with numerous processes, and by the appearance of a cobweb covering a large extent of the fundus. These are translucent at certain points, and extend freely in the vitreous. They have pedicles, polymorphous masses, irregular surfaces, marked prominences. They are located in any part of retina. Foci of pigment are rather frequent. They are often complicated by retinal detachment produced by traction of bands.

Traumatic proliferating chorioretinitis is always consecutive to a hemorrhage and to a rupture of the inner membranes. The fibrous patch is of more regular thickness, and is opaque everywhere. It does not extend into vitreous, but only proximate to it. It has a smooth and even surface without marked prominence. It is located more frequently in the macula, disc, and adjoining parts. It is not accompanied by detachment following the proliferation; on the contrary the latter binds the retina to the choroid. Foci of pigmentation are very frequent.

After a review of the literature, La Ferla believes that hemorrhage, usually from trauma, is probably always necessary to the production of the fibrinous bands.

Dimmer states that shot injuries with choroidal rupture were first examined anatomically by Ammon in 1855, then by Waldeyer in the case of Conn, later by Goldzieher, Ginsberg, Wagemann and his pupil Hermann, and finally by v. Szily, Igersheimer and Meller.

A. Fuchs reports a case of retinitis sclopetaria in which the retina and pigment epithelium became partly necrotic; the necrosed parts are absorbed and the surrounding tissues react with a proliferation, leading to the formation of cicatricial membranes. The new formed tissue develops from the choroid. It it partly connective tissue, and partly neuroglia from surviving elements of the retina.

Bachstez reports a case of a boy, age 15, shot in left temple, with exit of bullet thru right temple. Atrophy of disc, hemorrhage, and white foci with pigmentary changes at that macula. Autopsy showed these white foci to be due to a necrosis of the retina.

Wallace and Neame state that there is a degeneration of retina and fibrous tissue formation of the retina, and more still between retina and choroid and around the pigment. They reach the conclusion that some of the fibrous tissue arose from inflamed choroid tissue which fused with the retina.

All authors seem to be in agreement that there was found, besides changes of the inner coats of the eye, newly formed connective tissue, which forms a thickening, is in part degenerated hyalin, and contains pigment cells and free pigment. The remaining portions of the retina are covered on both sides by scar tissue. Retina and choroid are in places united by scar tissue into one coat, in which lumps of pigment are found. The remaining portions of the retina contain pigment which probably is derived from the pigment epithelium.

At the edge of retina-choroidal ruptures, retinal duplications were found. These may extend over the optic nervehead and manifest themselves by a contortion of the vessels and formation of vascular curves. Meller emphasizes the traumatic necrosis of retina and choroid, both of which are then supplanted by the thickening scarring process.

Diagnosis of chorioretinitis proliferans traumatica is generally not difficult. The type of trauma usually points to the correct interpretation of these finds. These findings may resemble classical proliferating retinitis, but in the latter there is no destruction of the choroid, and the pigmentations are much less

prominent. In exudative retinitis, the white sections are always below the retinal vessels, and no choroidal changes or pigmentations are visible.

Dimmer and Pillat, in their Atlas, show a number of photographs of this condition.

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DISCUSSION.

DR. CHARLES A. BAHN, New Orleans, La.: Dr. Weih's interesting case forcibly illustrates how much damage can be done to a pair of eyes by a bullet, without its actually touching either one of them. We have here a concussion injury to both eyes, with section of the optic nerve and evulsion of the retinal vessels in the left eye. The tremendous force of the bullet, with its forward, rotary and spiral movement thru the orbit, was transmitted to the posterior eyeball with the result you have seen. Another type of concussion injury seen during the World War was cataract produced by proximity, but without actual contact with an exploding shell.

In the reparative process, the dead tissue was absorbed and lymph and fibrin invaded the retina and choroid, with attendant fibroblast deposit, which in time produced the greater part of the newly formed tissue. The blood was absorbed, with or without pigment being left behind, and there was also a proliferation of the glial and pigment cells. Because of these many variables, no two cases of this condition are exactly alike.

Altho extensive bleeding is usually a prominent factor in the proliferative process, because of its slow absorption, especially in a disorganized vitreous, which means increased fibroblast deposit, we must not lose sight of the fact that certain normal and abnormal constitutents of the blood and lymph partially regulate scar tissue formation, especially following injury. Thus, in persons with diabetes and tuberculosis, the tendency to scar tissue formation is illustrated in numerous intra- and extraocular symptoms of these diseases.

DR. ARTHUR J. BEDELL, Albany, N. Y. A few photographs are shown to illustrate traumatic proliferating retinochoroiditis. Mrs. M. K. was examined in 1919 and found to have normal vision in each eye. Her fundi were without pathologic change. In 1926, she complained of poor sight in her right eye and stated that she had been in an automobile accident in 1924, at which time the right malar region was broken. She had no scars on her face or on the eyeball.

The first picture was taken on the 25th of February, 1927, and, as here illustrated, shows a large white mass overlaying the upper portion of

the disc. The outer half of the nervehead is well outlined. The veins are straight, the arteries small, in parts thread like, with white walls and narrow lumen. To the temporal side of the macula, there are many white spots in the retina. The proliferating mass was photographed, and is shown in the second slide as a fluffy mass in which a group of newly formed blood vessels pass forward to the apex of the elevated connective tissue formation. The third picture of the same patient illustrates an isolated view of a portion of the periphery of the fundus, the obliterated arteries are seen as white streaks, the lumen of the veins is unequal, with exudate along the course of some of them, and white spots lying between the vessel divisions—a typical case of retinitis proliferans without penetration of the eyeball.

This was contrasted by two other photographs, one showing large starlike proliferations of elevated white tissue with marked vascularization. The other was a proliferation of newly formed connective tissue which extended from the disc obliquely down to a large patch of destroyed choroid and retina. In this case, the striations in the wrinkled retina are very clearly demonstrated. The artery changes are less extensive than in the first patient.

Dr. E. P. Weih, Clinton, Ia. (closing): I wish to thank Dr. Bahn for bringing out these additional points. I also wish to emphasize the fact that our case was due to concussion, not to direct injury, and that the damage that results at first is very much more than we think.

Just a word about the pathology. All authors seem to be in agreement that there was found, besides changes of the inner coats of the eye, newly formed connective tissue, which forms a thickening, is in part degenerated hyalin, and contains pigment cells and free pigment. The remaining portions of the retina are covered on both sides by scar tissue. Retina and choroid are in places united by scar tissue into one coat, in which lumps of pigment are found. The remaining portions of the retina contain pigment which probably is derived from the pigment epithelium.

At the edge of retinochoroidal ruptures, retinal duplications were found. These may extend over the optic nervehead and manifest themselves by a contortion of the vessels and formation of vascular curves. Meller emphasizes the traumatic necrosis of retina and choroid, both of which are then supplanted by the thickening scarring process.

Diagnosis of chorioretinitis proliferans traumatica is generally not difficult. The type of trauma usually points to the correct interpretation of these findings. These findings may resemble classical proliferating retinitis, but in the latter there is no destruction of the choroid, and the pigmentations are much less prominent. In exudative retinitis, the white sections are always below the retinal vessels, and no choroidal changes or pigmentations are visible.

RETINAL HEMORRHAGE AS AN EXPRESSION OF VICARIOUS MENSTRUATION.

CLARENCE LOEB, A.M., M.D., and S. J. MEYER, M.D.

CHICAGO, ILL.

By vicarious menstruation is meant an extragenital hemorrhage at or near the time of normal menstruation, either substitutional for it or complemental to it. This phenomenon occurs very rarely, Roth[18] having been able to collect only 225 cases from the literature since 1870. The organs and tissues affected, in order of frequency, are: the nose (Walker,[21] Coughlin,[4] Macht[15]), stomach (Kuttner,[14] Darnall[5]), intestinal canal (Holmes[12]), lungs (Ford[8]), mammary gland (Novak,[18] Thornton,[20] Hirschberg,[11] Ziegenspeck,[18] Lambinon,[18] Condit[3]), skin (Parrot,[18] Mueller,[18] Chambers,[18] Anderson,[18] Opel[18]), lips (Hauptmann,[10] Coughlin[4]), eye (Huizinga,[18] Powell,[18] Claiborne,[18] Meanor,[16] Espino,[7] De Schweinitz,[6] Terrien and Cousin[19]), nevi (Bloom,[2] Condit[3]), kidneys (Ford,[8] Cuturi,[18] Dschigit[18]), old cicatrices (Kerley[13]), abdominal fistulae (Bircher[1]), umbilicus (Gardner,[9] Cullen[18]), gums (Beers[18]), stumps of amputated extremities (Puesch[18]).

There are several explanations of vicarious menstruation, the most logical of which is based upon a theory which tries to explain normal menstruation. This theory states, that there is in vicarious menstruation, in the tissues of the affected area, an abnormal sensitiveness to the ovarian hormones, analogous to the normal sensitiveness of the endometrium in normal menstruation.

Disorders of menstruation are frequently associated with ocular lesions. De Schweinitz[6] assigns such an etiology to vitreous opacities, hemorrhagic retinitis, optic neuritis and primary optic atrophy, Terrien and Cousin[19] mention the following conditions as being dependent on menstrual disturbances: conjunctival hyperemia, hemorrhages into the retina and vitreous, diminution of visual acuity, accommodative asthenopia, contracted visual fields, crises affecting the trifacial which lead to increased intraocular tension and even glaucoma, photopsia, hyperemia of the

disc, amaurosis, and edema of the lids. Espino[7] reported the case of a woman who always had a hyperemia of the conjunctiva at the time of menstruation, while Meanor[16] described the case of a woman who at each menstrual period had a hemorrhage into the vitreous, the opacities lasting nearly to the next period. She had had blurred vision during menstruation ever since its inception. He also spoke of a case of sarcoma of the iris which had a hemorrhage into the anterior chamber at each menstrual period. In the discussion, Van Kirk[16] spoke of a woman who had an edema of the eyelids and cheeks for three or four days at each period, and reported also the case of a woman who developed two or three blebs on the eyelid when she began to menstruate. Huizinga[18] reported the case of a girl of 17 who had an attack of dizziness and faintness, followed by a rapidly developing dimness and distortion of vision, first in the left eye, and in a few minutes in the right. The fundi showed the picture of hemorrhagic retinitis. The spots cleared up after the menstruation, only to recur at two subsequent periods, while she was under observation.

The patient whose ocular condition was the incentive to the preparation of this paper, is Miss M. S., white, aged 26, occupation—social worker. She was first seen at the Michael Reese Dispensary, on Sept. 24, 1927. Her complaint was headaches, recurring about once a month for the past year, which seemed to be worse following the use of the eyes, but which were unaffected by the state of the weather. Between these attacks, she felt well. The periodicity of the attacks led to an inquiry about their relation to the time of her menses. The patient was at first uncertain about this, but on subsequent visits rather inclined to the belief that the time relation was intimate.

Externally, her eyes were normal. Manifest refraction showed an emmetropia in each eye. With both eyes open, could read the 6/6 line with difficulty when plus 0.5S. was placed in front of each. The fundus of the right eye was normal. In the left eye, just where the upper horizontal temporal branch of the arteria centralis retinae leaves the disc, there was a flame shaped hemorrhage, about 1 d.d. in length, and 1/3 d.d. in height at its broadest part. In the temporal periphery, were a couple of isolated, small, black spots, each surrounded by a light reddish area, giving the impression of their being remnants of previous hemorrhages. The remainder of the fundus showed nothing abnormal.

The family history of the patient revealed that an uncle and a cousin had died of tuberculosis. The personal history contained nothing significant except the statement that her menses were always delayed and painful. The physical examination revealed nothing abnormal except a small cyst of the right ovary. All laboratory tests were normal. No treatment instituted.

Oct. 1, one week later; hemorrhage almost absorbed.

Oct. 15, the fundus shows no sign of the hemorrhage formerly present.

Oct. 19, states that her menses were on the 17th, at which time she had had occipital headache, but no subjective ocular symptoms. Both fundi were normal, ophthalmoscopically; V=6/6.

The patient was seen at irregular intervals up to December, at which time, the illness of one of us (Loeb), who had been following the case, was followed by the disappearance of the case from observation. Altho the headaches recurred, no other hemorrhage was ever observed.

The authors recognize the fact, that the diagnosis of retinal hemorrhage due to vicarious menstruation is open to objection, but when we take into consideration the history of the periodic monthly headaches, of short duration, occuring about the time of menstruation, which, itself, was always delayed and painful, coupled with normal refraction, normal physical findings and normal personal history, we feel that there is a causal relationship between the evidently abnormal menses, the presence of blood in the retinal tissue and the evidence of previous retinal hemorrhages, sufficient to justify the diagnosis of retinal hemorrhage due to vicarious menstruation.

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DISCUSSION.

DR. WILLIAM H. CRISP, Denver, Colo.: In my eye practice, I have had no personal experience with any such cases as are referred to in this general classification. I happened to have had a young woman relative who used to develope epistaxis rather frequently about the time of menstruation, and I think there is some suggestion that hemoptysis may occur as a complication of this kind.

I believe the term "vicarious menstruation" is quite often very loosely applied, and strictly speaking perhaps is rather unscientific. The suggestion would be that Nature, normally preparing for a hemorrhagic process in one organ of the body for a fairly prolonged period, once a month, during the active sexual life of a woman, sometimes substitutes a like process in another organ. In most of these cases, there does not seem to be an exact justification of the term. In a few cases reported, there was amenorrhea with hemorrhage in the eye, but in a number of cases, it seems to me the connection is rather vague and uncertain. We must remember that with a great number of women, perhaps 20 per cent of the whole time, for a good many years, is occupied in the menstrual process. It would not take much of a coincidence to bring a good many of these accidents within that period. With any profound disturbance in the body, it seems probable that the permeability of the vessels is altered, and the chemistry of the blood is more or less altered at that time. Further, we must remember that there are a good many obscure cases of ocular tuberculosis associated with hemorrhage, and it seems likely that some of the cases reported as vicarious menstruation in the eye have been of this character, the general disturbance of the whole system inducing or aggravating bleeding from the retinal vesesls at this particular time.

DR. D. L. TILDERQUIST, Duluth, Minn.: Unless it be an actual reporting of the facts of the case, any discussion of this topic must be, as Dr. Crisp has stated, largely speculative. He also mentioned that during the menstrual period there must be changes of some nature in the general metabolism, or in the general vascular system, or in some other way. So far as I can find from the literature I was not able to determine what the changes are. There apparently is very little known concerning any general changes of metabolism during the menstrual period. It is not that there is lack of literature. For instance, one investigator made a great many examinations of the blood in a selected group of women during the menstrual period and during the intermenstrual period, with a view of determining whether there was any variation of calcium content of the blood. His conclusions are, that there is a rise of calcium content during the menstrual period. Another group of investigators made

similar findings as to the lipoid content of the blood. Both groups concluded that there are very few variations in the blood content, and both groups added that there were so many factors in the ordinary life of the individuals examined which they could not gauge or control, and which might affect the results, that, therefore, their conclusions were in no sense reliable.

It seems to me that until these fundamental changes during the menstrual period are known, we cannot differentiate hemorrhages of the socalled vicarious nature from hemorrhages due to pathology.

Dr. Ray K. Daily, Houston, Tex.: I think this history might be interesting in this connection. A woman in the early thirties suddenly discovered that she could not see out of her left eye. She consulted an oculist, who found that she had a hemorrhage at the macula. Four years later, her right eye became inflamed during a menstrual period, and ever since she has recurrent inflammations in the right eye, always occurring at the menstrual period, and subsiding as the menstrual period passes. This eye shows evidence of blephritis, conjunctivitis, and iritis. An examination by an internist was negative for constitutional disease or focal infection. In view of this history, the hemorrhage may be considered as a form of vicarious menstruation.

Dr. S. J. Meyer, Chicago, Ill., (closing): As Dr. Crisp said, it might be well to rule out tuberculosis, which is sometimes hard to do. In this case, there was a history that the patient had been suspected of tuberculosis, altho she had not shown any signs.

I just had a note from Dr. Alvis, who states that he saw a colored woman, aged thirty, who had profuse subconjunctival hemorrhage at one of her periods, which had occurred at several previous periods.

I have been watching a patient for the last few months, a young woman thirty-one years old, who revealed the first time she came to the office the socalled pink eye, which turned out to be a marked hyperemia of the conjunctival vessels. She stated that this was the second time it had appeared, once about a month before. I followed this case up, and found that each time this occurred it was during her menstrual period. No treatment was given, and I had her come back a week later after each period, and the eye was in much better condition.

THE FORMATION OF THE AQUEOUS HUMOR, ITS RELATION TO INTRAOCULAR AND VASCULAR PRESSURES.*

ARTHUR M. YUDKIN, M.D.

NEW HAVEN, CONN.

More than ordinary importance should be attached to accurate information regarding the composition of the intraocular fluid, because such knowledge must be depended upon to furnish a clue regarding the origin and alterations thereof. The much debated question, as to whether the aqueous humor represents a specific secretion by a special structure such as the ciliary body and processes, or merely a fluid having its origin thru relatively simple physical and physicochemical processes from the circulating fluids of the body—the blood and lymph—hinges upon the comparison of the composition of the various fluids involved and a consideration of the forces by which the production of the ocular fluid is accomplished.

Several investigators have studied some of the more important components, such as the sugar, the protein and the chlorid, but the other constituents have scarcely been touched upon. No one species of animal has been utilized consistently, hence the compilation of comparative results has been almost impossible. This is not the place in which to present a detailed critique of the shortcomings of the earlier experiments. Their results show a lack of agreement with respect to the quantitative occurrence of most of the constituents. Many of the data involved have recently been summarized by Duke-Elder[1] and others.

It has been our purpose to determine with analytic precision the composition of the normal intraocular fluid of living animals and compare the findings, where possible, with those for their

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*The expense of this experimentation was defrayed in part by a grant from the Committee on Scientific Research of the American Medical Association.

blood and spinal fluid. All the analyses were made on single specimens of the aqueous humor withdrawn from the anterior chamber of the eye. We were confronted with the difficulty that other workers have been obliged to face, namely, the small quantity and minute concentration of some of the substances in the aqueous humor. The average values secured in our laboratory for the constituents of this fluid, the spinal fluid and the serum of the dog are summarized in Table 1. These findings compare favorably with those determined by others on the same species.

TABLE I.

	Milligrams per 100 c.c. of fluid.		
	Intraocular fluid	Spinal fluid	Serum
Glucose	91.9	85.1	79.1
Sodium Chlorid	632.	508.	497.
Total nitrogen	35.2	36.4	.....
Nonprotein nitrogen	28.6	29.1	33.8
Protein nitrogen	6.6	7.5	.....
Amino acid nitrogen	9.1		
Urea nitrogen	12.1		
Uric acid	1.7	1.7	1.7
Inorganic sulphat (determined as SO_4)	0.36		
Inorganic phosphat (determined as P_2O_5)	1.1		
Refractive index	1.33516	1.33512	.....
Volumes percent CO_2 per 100 c.c.	61.7	52.	46.3

From the results of the analyses recorded above, it may be noted that, so far as all the major constituents are concerned, the composition of the aqueous humor in the dog has a striking resemblance to that of the serum and cerebrospinal fluid. The chemical composition of the ocular fluid appears to be nearly identical with that of the cerebrospinal fluid. The aqueous humor reveals a small amount of protein when compared with serum. The sodium chlorid concentration, on the other hand, is considerably higher in the aqueous humor than the blood serum.

Lehmann,[2] Duke-Elder,[1] Cohen,[3] Wittgenstein and Gaedertz[4] showed that the sodium, potassium, calcium and magnesium ion concentration in the aqueous humor is less than that in the blood serum, and that the chlorid, phosphorus and sulphur ion concentration is greater in the aqueous humor than in the blood serum. It appears, therefore, that there is a difference in the ionized constituents of the fluids. The cations are present in less concentration, and the anions in greater concentration in the aqueous humor than in the serum. The remaining sub-

stances found in the blood serum are distributed almost equally in both fluids. The constituents of the fluids may be divided into two groups, according to the characteristics of the molecules in solution. To the first group of slightly diffusible compounds belong such ingredients as the proteins; the second group includes the readily diffusible substances which are distributed in the plasma and aqueous humor. The latter group may again be divided into nondissociated and dissociated diffusible substances.

Mestrezat,[5] Van der Hoeve[6] and Osborne,[7] employing the method of depression of the freezing point, found the aqueous humor and the blood serum essentially isotonic; and Duke-Elder,[1] by means of a microosmometer, noted that it corresponds to that of a dialysate of the blood in the arterial capillaries. The specific gravity of the aqueous humor of the dog has been determined as varying between 1.007 and 1.009, and the refractive index between 1.3335 and 1.3360. The surface tension estimated by Dieter,[8] was 72.4 to 73.0 dynes/cm, at 18° C. The viscosity of the aqueous humor in the dog was found by Scalini[9] to be greater than that of water and less than that of the blood. The conductivity of the aqueous humor was found to be greater than serum. Van der Hoeve[6] found the conductivity to be the mean between the dialysate of arterial and venous blood. The hydrogen ion concentration was estimated to be pH 7.40-7.50, which is slightly higher than blood serum. It appears therefore, that the normal aqueous humor is a product of the blood, and is separated from it thru a membrane which is relatively impermeable to large sized molecules. Those constituents which are ionized are partitioned variably in the two fluids, whereas the nonionized diffusible substances are found in equivalent proportions.

After the foregoing consideration of the composition and physical properties of the aqueous humor and its comparison with other fluids of the body, a presentation of the evidence supporting the various hypotheses may lead to a better understanding of the probable formation of the intraocular fluid.

Secretion Theory.

The supporters of this theory have considered the ciliary body and processes as the chief source of formation of the intraocular fluid. The functions of these eye tissues have been likened to that of the choroid plexus of the cerebrospinal sys-

tem, because of their anatomic structure and cellular performance. The ciliary body and its processes were regarded as a gland by early observers. Nicati[10] and others described the epithelial layer of these structures as glandular in nature. Boucheron[11] considered the anterior part of this area as secreting the aqueous humor, and the posterior part as secreting the vitreous. Later, Collins[12] described epithelial outgrowth covering the ciliary body and processes as ciliary glands. These findings are not in accord with the histologic studies presented more recently by Finnoff,[13] Salzmann[14] and others.

Seidel and Schmelzer[15] observed cytologic changes in the epithelial covering of the ciliary processes following cellular activity, which were considered by them as manifestations of glandular processes. They found an increase in the size of the cell, which also differentiated itself into two zones: a granular area at the inner border, and a clear zone at the periphery or free margin of the cell. Hemosiderin granules and mitochondria were also observed in the tissues. The same cellular disturbance was noted in the choroid plexus under similar conditions. Becht[16] pointed out that in the latter the above changes in the cells did not constitute evidence of active secretion, and the alteration of cellular tissue was the exact opposite of that which occurred during active secretion in typical glands. Weed[17] also demonstrated that the intravenous injection of distilled water caused analogous changes in the choroid plexus. He interpreted the alteration in the cells as evidence of an increased amount of fluid passing thru them.

Further evidence of active secretion has been sought by Nicati,[10] Seidel[15] and others, by introducing into the general circulation, and locally into the cul-de-sac, such agents as pilocarpin and physostigmin, which produce true secretion in other glands of the body. Changes were noted in the cellular tissue as described above, and the manifestation was accompanied by an increased protein content in the aqueous humor. The reactions to the miotics were in accord with those set forth by Wessely,[18] Yudkin[19] and his coworkers, but the above finding cannot be interpreted as necessarily indicative of glandular activity. This was also upheld by Koller[20] and Thiel,[21] who produced evidence to show that physostigmin caused a vasodilation in the ocular tissue; and Adler and Landis[22] demonstrated, that even tho the ciliary ganglion was severed or degenerated, the miotics produced the same effect and hence the action must be

vascular in origin, rather than due to glandular activity. It was shown that atropin had the opposite effect on the vascular tissue.

Seidel[15] further supported his secretory theory by showing that there is a difference of electric potential between the ciliary body and processes and the cornea, so that a current can be established as in the case of a secretory structure like the salivary glands; but this manifestation was questioned by Lullies and Gulkowitsch[23] and Duke-Elder.[1] They believed that this electrical phenomenon might be explained by the fact that the two electrodes which Seidel[15] used were immersed in solutions of different ionic concentration.

It is a known fact, that in the active secretion of a gland, there is usually some substance newly formed within the cell for some specific purpose in the body. For example, the salivary and pancreatic secretion contain enzymes not found in the blood, and which are necessarily manufactured de novo within the glandular structures. So far as I know, substances not occurring in the blood have not been detected in the intraocular fluid. In view of the fact that many authorities question the action of the miotics, pilocarpin and physostigmin, and the cellular changes produced by them and other means, as evidence of true secretion, and that the ciliary body and processes do not resemble other glandular tissue histologically, it is necessary to seek an explanation for the composition of the intraocular fluid by some other method than true glandular secretion.

Filtration Theory.

In opposition to the theory of physiologic secretion is that of filtration, which was advocated by Leber[24] and his school. They believed that the intraocular fluid is produced in the ciliary processes by a process of physical filtration, the principal factor in this performance being the gradient pressure from the source of origin to the point of absorption. This assumption was supported, in the main, by the experimental work of Henderson and Starling,[25] who showed that the production of intraocular fluid is strictly proportional to the difference of pressure between the blood in the capillaries of the eyeball and the intraocular fluid. They believed that the difference of pressure amounting to over 30 mm. of mercury is sufficient to account for the low protein content in the intraocular fluid, without

assuming an active intervention on the part of the capillary walls or of the ciliary body.

Our knowledge of the factors involved in fluid exchange has been considerably modified and augmented in recent years. All experimental work on this subject, however, reveals that the capillary membrane ordinarily is not permeable to the proteins of the blood plasma which circulate thru its lumen, and the membrane permeability varies in different species of animals and in different parts of the body in the same individual. It has been shown that the plasma proteins, unable to leave the capillaries, produce an osmotic pressure which tends to retain the fluid in the blood vessels, and that the capillary blood pressure acts in reverse manner. Krogh[26] demonstrated that the capillaries constrict and dilate independently of the arterioles, and that their caliber when the tissue they supply is at rest, may be much less than when the tissue is active. This idea was not in accord with the older belief, that with the heart functioning normally, the extent of the capillary circulation depended primarily on the contraction and dilation of the arterioles and secondarily on the venous pressure. Recently Landis,[27] utilizing delicate experimental technic, showed that the dilated capillaries are not more permeable than those of the constricted vessels, and that an increase of capillary pressure would cause water to pass toward the tissues, while a converse relation would lead to the movement of fluid into the blood. He also demonstrated that an injury of the capillary endothelium increases its permeability to protein, with a corresponding reduction of the colloid osmotic pressure. He believed, therefore, that the direction and the amount of water thru the normal capillary wall is determined primarily by the level of capillary pressure in association with osmotic forces.

Altho the present knowledge of capillary permeability may account for some of the ocular findings, there is still further explanation necessary, in view of the more recent contributions concerning the chemical content and physical properties of the aqueous humor. It was noted above, that the aqueous humor possesses considerably less protein than the serum, that the sodium chlorid content is greater in the aqueous humor than in the serum, that the undissociated diffusible crystalloids, which are largely represented by glucose, appear to be present in both systems in apparently equal concentration, and that of the dissociated diffusible crystalloids, the cations Na, K, Ca, and Mg

are in less concentration, and the anions Cl, PO_4 and SO_4 in greater concentration, in the aqueous humor than in the serum.

A critical review reveals that Mestrezat[5] observed a parallel difference in the concentrations of individual substances in the cerebrospinal fluid and the blood serum, and that the osmotic pressures of the two fluids are approximately isotonic, when measured by the depressions of the freezing points. By further investigation, he found the aqueous humor, like the cerebrospinal fluid, is an equilibrated dialysate of the blood serum. Meesmann and Baurmann[28] observed a similar interchange of the chemical constituents in the blood serum and aqueous humor. Freemont-Smith[29] also noted this physicochemical arrangement. He was able to show that a relationship exists between the concentration of plasma proteins and the distribution of chlorides, so that the greater the protein content of the plasma, the greater the excess of chlorides in the cerebrospinal fluid. He found that an analogous condition prevails between plasma and pleural, ascitic and synovial effusions respectively. It therefore appears that the formation of the aqueous humor must be accounted for in some other way than by the secretory process or the simple pressure filtration.

Recently a different view was proposed by Duke-Elder,[1] Meesmann,[2] Baurmann,[28] Magitot[5] and others. They believe that the unequal distribution of the ions in the two related fluids, serum and intraocular fluid, is characteristic of a system in membrane equilibrium by virtue of their relationship. Duke-Elder[1] champions the membrane equilibrium theory enunciated by Donnan[30] as applicable to the conditions of the fluid formation in the eye. Not only does the normal fluid obey this theory, but he found that the abnormal aqueous humor likewise underly the Donnan membrane equilibrium. He considers the capillary walls and not the ciliary epithelium as the essential dialysing membrane, because the intraocular fluids are in equilibrium with the capillary blood, the properties of the aqueous humor varied directly with the state of their permeability, and the ciliary region was not the only site of the process of dialysis. In such a system, he postulates "that there is a continuous process of reciprocal interchanges on either side of the capillary walls, made up of filtration outwards, determined by the differences in the hydrostatic pressures, an osmotic flow in the opposite direction, conditioned by the relative osmotic pressures, a diffusion out or in of the diffusible substances according to their

several differences in concentration and a readjustment depending on colloid impermeabilities, and that on either side of the capillary walls there is a balancing hydrostatic and osmotic pressure equilibrium around which level the mutually compensating pressures are continually fluctuating, and in any capillary these balancing forces will determine a flow of fluid outward at one movement and inwards at another." Theoretically the fluid is stagnant, but superimposed upon this primary metabolic interchange, there probably is a secondary and intermittent circulation, which is determined by the contraction of the internal and external musculature, the influence of the pulse beat, etc.

Comment.

From a chemical point of view, our findings of the normal and abnormal compositions of the intraocular fluid are in accord with those presented by the advocates of the membrane equilibrium theory. This agreement, alone, is not sufficient for us to accept the hypothesis proposed by Duke-Elder,[1] for other factors must be considered also. It has been shown that where a membrane is more permeable to one ion than another, a variable electric potential and chemical reaction exists in the separated fluids, and the osmotic and hydrostatic forces are equalized. This has been established by the experimental work of Mestrezat[5] and others, with colloidal sacs of known permeability. A critical study, however, reveals that between the capillary blood and intraocular fluid in vivo, it is impossible with our present technic to demonstrate the existence of parallel thermodynamic forces. As yet, no direct method except the microosmometer of Duke-Elder[1] has been devised for measuring these forces in the blood and the eye fluid of the same animal under normal physiologic conditions.

There are, however, certain changes, chemical and physiologic, which take place in the eye when the composition of the blood is altered and the permeability of the capillaries is changed, which point toward the theory of membrane equilibrium. Certain disturbances of the osmotic and hydrostatic pressures of the two systems produce changes which may be accounted for more readily by the assumption of this hypothesis. A review of these observations may help us understand the situation better.

Duke-Elder[1] injected large quantities of gum arabic into the blood of cats and found no change in the refractive index. Neame and Webster,[31] Yudkin[19] and his coworkers, demon-

strated that arsenic, even tho injected intravenously, did not appear in the intraocular fluid until the permeability of the vessels was altered. Gaedertz and Wittgenstein[4] showed that intravenously injected diffusible basic dyes and inorganic cations cannot be recovered in the normal aqueous humor under ordinary physiologic conditions. The situation is reversed for diffusible acid dyes and anions. This behavior is attributed to the protoplasmic absorption of the substances. The cathodal substances are absorbed rapidly from the blood stream, and the amount required for transmission to the aqueous humor is enormous. In large amounts they are exceptionally toxic. The anodal substances, on the other hand, are absorbed very poorly, and hence accumulate to a greater extent in the blood stream, and therefore less of the ingredient is necessary for transmission to the intraocular fluid. From this it is concluded that the diffusible electrolyte depends for its transport on the function of its electric charge. Van Creveld and de Haan[32] showed that intravenously injected fluorescin was present in the aqueous humor in exactly the same concentration relative to the plasma as they obtained by dialysing blood thru colloidion membranes. It has likewise been shown that iodid, given by mouth, subcutaneously, subconjunctivally and intravenously, finds its way into the intraocular fluids, and that its concentration in the aqueous humor varies with that of the blood. Bromid and salicylat have also been recovered in the aqueous humor after administration by mouth.

With disturbances of carbohydrat metabolism resulting in either hypoglycemia or hyperglycemia, fairly comparable alterations in the concentration of glucose in the aqueous humor are exhibited. We have noticed, however, that in either extreme, the sugar content is greater in the aqueous humor than in the blood. It therefore appears, that when the concentration of colloidal substances in the blood is altered, their transmission into the intraocular fluid is not apparent and is probably due to their large sized molecules. On the other hand, it seems that diffusible substances align themselves according to (a) their affinity for plasma proteins and (b) their ability to dissociate into basic and acid radicles. Nondissociable diffusible ingredients of the blood plasma are partitioned relatively equally on both sides of the membrane.

It has also been shown by Duke-Elder,[1] Yudkin[19] and others, that where the permeability of the capillary walls is altered by

(a) lowering the intraocular pressure either by a paracentesis of the anterior chamber, or by removal of the vitreous humor, (b) local irritation produced by mechanical manipulation or subconjunctival injections of chemicals, (c) stimulation of the cervical sympathetic and, (d) radiant energy (Duke-Elder), the composition of the intraocular fluid is considerably modified. The product of increased permeability has a greater amount of protein, a decreased concentration of salt, while the nonionized diffusible substances have an apparently equal distribution in both fluids. Duke-Elder[1] also notes that the anions relatively decrease and the cations increase with the increase in the concentration of colloids, so that the altered intraocular fluid appears to be formed by the same process which produces the normal fluid. From the above observation it may be seen that the intraocular fluid may be altered by changes (1) in the composition of the plasma and (2) in the permeability of the capillary wall.

It has likewise been adequately demonstrated clinically and experimentally that the intraocular pressure may be altered in several ways. By experiment it has been shown that the intraocular pressure varies directly with that of the blood. As yet, this parallel relationship between the arterial pressure and intraocular pressure has not been verified clinically by most ophthalmologists. It has been demonstrated, however, that the intraocular pressure varies very closely with the venous pressure. Obstruction of the vortex veins as they leave the sclera increases the intraocular pressure. Even a ligature on a single vein produces a similar but less marked increase. Carl Ludwig, long ago, pointed out that capillary pressure was far more dependent on venous pressure than on arterial pressure. This observation is confirmed by Carrier and Rehberg[33] on capillary pressure in the hand. The intraocular pressure may also be influenced by injections of anisotonic substances into the blood stream. When hypotonic saline solution is used, the pressure in the eye rises synchronously at first with that of the blood pressure, and soon drops simultaneously with it. Later, it continues to rise independently of the blood pressure, and remains elevated for a considerable time. On the other hand, a hypertonic solution produces an initial fall in the intraocular pressure, which subsequently continues to rise slightly, almost parallel with the pressure in the blood. When the injection ceases, the intraocular pressure begins to fall without a similar decrease in the

blood pressure. The injection of isotonic fluid apparently does not alter the normal status of the intraocular pressure. Hertel,[34] Duke-Elder[1] and others show that the increased intraocular pressure of glaucoma may be lowered by hypertonic solutions injected into the blood stream. Corresponding results are obtained experimentally by Freemont-Smith[29] and his coworkers in the cerebrospinal fluid. Clinical corroboration of lowering of the intracranial pressure is recorded by Cushing[35] and his coworkers.

In order to understand the changes which take place in the intraocular pressure, some knowledge of the vascular pressure is necessary. Altho several observers have estimated the vascular pressures in the eye, no one as yet has succeeded in presenting a technic which is flawless. The direct method proposed by Duke-Elder[1] appears to have many favorable possibilities, but is still experimentally impracticable for all eyes. He calculates the mean pressure in the ophthalmic artery in the cat to be about 100 mm. mercury, and in the retinal artery about 75 mm. mercury. The pressure in the venous exits in the dog is normally slightly above the intraocular pressure. This venous observation is however, contrary to most experimental reports. From his estimates he suggests that the capillary pressure in the eye varies from a few mm. mercury above the intraocular pressure to a height of about 50-55 mm. mercury.

Résumé.

A recapitulation of the facts shows that the intraocular fluid is derived from the blood stream by the same forces that are responsible for other tissue fluids. The factors involved in the formation of the fluid are the hydrostatic pressure of the two systems, the permeability of the intraocular vascular capillaries and the osmotic pressures of the blood plasma and the intraocular fluid. The authorities who believe that the aqueous humor is produced as a pressure filtrate base their assumption on experimental work which shows that the hydrostatic pressure in the intraocular vascular bed is a gradient one, i. e., the ciliary capillary pressure is greater than the intraocular pressure, which in turn is greater than the pressure in the veins and canal of Schlemm. The composition of aqueous humor may also be accounted for by the difference in the pressures of the blood stream and the intraocular pressure. Because of this change in the hydrostatic pressure from the site of origin to the exit

of the fluid, there is a continuous circulation present. The intraocular pressure is considered as a function of the intraocular vascular hydrostatic pressure, and it rises and falls with the blood pressure. Some lay stress on the arterial influence and others on the venous system. The normal intraocular pressure therefore represents the pressure at which the rate of formation of intraocular fluid is exactly balanced by the rate of escape from the eyeball.

The pressure filtration theory does not account for the chemical composition of the aqueous humor under either normal or abnormal conditions. If no other factors than filtration are involved in the separation of aqueous humor from the blood, one would expect the composition of the filtrable material to be identical in both fluids. It is known, however, that the proteins are present in the normal aqueous humor only in traces, and when the permeability of the membrane is altered, their proportions vary according to the laws of diffusion. The diffusible ingredients partition themselves in the fluids according to their electric potential in the normal and likewise in the altered state. The nondissociable constituents are partitioned almost equally on both sides of the membrane. The physical forces are not as well known as the chemical composition of the fluids, but the changes which present themselves when the hydrostatic and osmotic forces are altered, point to the fact that the pressure filtration theory needs modifications.

Conclusion.

It is my impression that the intraocular fluid is a dialysate of the blood plasma, in the sense that there exists a membrane —capillary wall and tissue covering it—which has a functional adaption to keep the media of the eye transparent and help regulate the slow continuous process of exchange—metabolic and catabolic—on both sides of the membrane, so that there is established a delicate balance between the hydrostatic and osmotic pressures of the arterial and venous plasmas within the eye. This balance is the intraocular pressure. There is a continuous circulation of the intraocular fluid in the eye. The rate of circulation depends on the metabolic exchange and the muscular reaction of the surrounding tissue.*

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DISCUSSION.

Dr. Harvey D. Lamb, St. Louis, Mo.: It is presumptuous, to say the least, to attempt to discuss Dr. Yudkin's important observations on the aqueous humor, not having engaged in any experimental work in this field. My own observations are confined to the paracenteses of rabbits' eyes and the noting, after enucleation, the formation of cystic swellings among the epithelia of the ciliary processes; enucleation was done at varying intervals from the time of the corneal puncture.

Since we may think our knowledge of the aqueous humor is altogether modern, I will mention the following facts.

The aqueous humor was known to the ancient Greeks as a thin colorless white-of-egg like fluid. Galen (131-201 A. D.) stated, that when the aqueous was too scanty, there occurred a drying up of the crystal or lens substance. Galen also knew of the regeneration of the aqueous after its loss thru a wound. St. Yves (1722) and Janin (1772) thought the aqueous humor came from the vitreous. Haller (1754), Zinn (1780), and others considered the source as being both surfaces of the iris and the ciliary body. Demours (1818), Descemet (1758), Wrisberg (1730-1808), Wardrop (1818) and others thought that a serous membrane lining the anterior and posterior chambers secreted this fluid, as in the case of the pleura, pericardium, arachnoid, peritoneum and joint linings. Arnold (1832) noted that when when the pupillary opening was closed by adhesions between the iris and lens the amount of aqueous in the anterior chamber was not diminished. Mery (1707) and Beer (1806) observed, that in closure of the pupil, the posterior chamber became swollen with aqueous.

Berzelius, in 1840, made the first chemical analysis of the aqueous of which we have record. Lohmeyer, in 1854, and Cahn, in 1881, gave the percentage amounts of eight and ten constituents of the aqueous. Many more chemical analyses of this fluid have been done with a great diversity of results.

Dr. Yudkin's very valuable contribution to the knowledge of the aqueous humor, as presented in this paper, can be divided into three parts. First, the report of the accurate quantitative determination of the principal inorganic and organic ingredients of the aqueous, spinal fluid and serum of the dog; second, a discussion of the method of formation of this important fluid; and third, the relation of the intraocular tension to the blood pressure in the bloodvessels of the eyeball.

Mestrezat and Magitot presented, in 1921, a table of the comparative amounts of many chemical constituents in the aqueous humor and the cerebrospinal fluid of the horse. In 1927, Duke-Elder made a similar detailed and extensive chemical analysis of the aqueous and blood serum in the horse. These findings were presented in his very excellent work "Recent Advances in Ophthalmology." Dr. Yudkin's comparative table of the amounts of many constituents in the aqueous, spinal fluid and blood serum, and the two others of Mestrezat-Magitot and Duke-Elder represent the high points in this work. These analytic tables must be compared in detail by those interested to determine the similarities and differences.

As Dr. Yudkin states, the problem of the formation of the intraocular fluid is the same problem as for the formation of the other tissue fluids of the body, that is of the lymph, the cerebrospinal fluid, that of the pleural, pericardial, peritoneal serous membranes, and that of the synovial membranes. Let us then examine what the general physiologists say about lymph production. Since in the text books, the other authorities generally refer to the work and opinion of Starling on this point, we will consult him. Starling states that the factors for the formation of lymph are mechanical and chemical; the mechanical depends upon blood pressure in the capillaries and the permeability of the capillaries to the proteins. The chemical factor depends chiefly on the metabolism of the cells outside the vessels. That whereas the formation of lymph in the intestines and liver can be explained by pressure filtration, in cases of the extremities and connective tissue generally, the chemical factor enters, and diffusion and osmosis explain the passage of water and dissolved substances. Every active contraction of a muscle is attended by the breaking down of a few large molecules into a number of smaller ones. The latter are discharged thru the semipermeable cell membrane into the surrounding tissue fluid, whose osmotic pressure is thereby increased. Water is accordingly drawn from the plasma of the blood capillaries, increasing the volume of the tissue fluid.

Dr. Yudkin has discussed every possible factor influencing the formation of the aqueous. His paper is worthy of much study, for it is from this basic experimental and theoretic field, that the pathogenesis of glaucoma will finally come. In a recent brilliant review of the pathology of glaucoma, Schmelzer states, that while we know today the mechanics of the increase of intraocular tension, we have little knowledge of the factors causing the primary glaucomatous eyeball to become relatively water tight and impermeable to the outlet of the aqueous humor.

Dr. Henry P. Wagener, Rochester, Minnesota: Unquestionably, the chemical and physical characteristics of the aqueous favor the view that the aqueous is a dialysate of the blood plasma in membrane equilibrium with the capillary blood. The opinions of Yudkin agree very closely with those of Duke-Elder, and there is a large amount of experimental evidence in support of their contentions. Perhaps the most persistent opponent of their ideas at the present time is Seidel of Heidelberg, who still believes that the aqueous is a secretion of the ciliary processes, a "simple secretion" as he terms it, in which no new substance is produced.

One of Seidel's strongest arguments against the filtration and dialysis theories is based on the question of hydrostatic pressure. Duke-Elder states: "To equilibrate the difference in osmotic pressures between the aqueous and the blood, the hydrostatic pressure in the capillaries should be from 25 to 30 mm. Hg. above that in the chambers of the eye." To support his contention that this condition exists, he advances, as his estimates of intraocular arterial pressure, the figures of the 84 mm. Hg. systolic and 64 mm. Hg. diastolic in the cat and 45 to 60 mm. Hg. diastolic in man. He believes, that the capillary pressure in man varies from a few mm. above the intraocular pressure to 50 or 55 mm. Hg. or more. In sharp contrast with these figures, stand those of Baillart and Magitot, who estimate the diastolic pressure in man as 30 to 35 mm. Hg. and the systolic as 65 to 70 mm. Hg., while the capillary pressure they believe to

lie between 25 and 30 mm. Hg. Seidel agrees with Baillart's figures, and supports them by measurements of the pressures in the anterior ciliary arteries and veins. Here he finds the arterial pressures to be, systolic, 55 to 75 mm. Hg., and diastolic, 35 to 45 mm. Hg., and the venous pressure to be 10 to 15 mm. Hg. He, therefore, estimates the capillary pressure to be about 25 mm. Hg. He believes, therefore, that the difference between the hydrostatic pressure in the ciliary capillaries and the intraocular pressure is too small to permit of the formation of the aqueous by filtration or dialysis. Baillart has recently published an article in which he warmly defends his figures against those of Duke-Elder. Clinically, the figures of Baillart seem to be more nearly correct.

Duke-Elder admits that the variations in the intraocular pressure produced experimentally by variations in the hydrostatic pressure in the circulation are not parallelled clinically in cases of vascular hypertension. The mechanism of the rise of blood pressure and the response of the capillaries in the two instances are entirely different.

Treacher Collins' contention that, anatomically, the ciliary processes are of glandular nature, does not seem to carry much weight, especially since it has been contradicted by other workers, as Yudkin points out.

From a clinical standpoint, with reference to the etiology of glaucoma and the present methods of its treatment, the dialysis theory of the origin of the aqueous would seem to be rather unsatisfactory. Both Collins and Seidel support the existence of an active circulation of the aqueous. The dialysis theory seems to necessitate a relatively stagnant aqueous except in the event of rather rapid and definite changes in the hydrogen-ion concentration or tonicity of the blood plasma. Neither Yudkin nor Duke-Elder believes that the aqueous is stagnant. Duke-Elder says, that as a result of thermal differences between the cornea and the iris, of the action of the extraocular muscles, and of the suction of the scleral spur, there is "an almost continuous, altho very minute, circulation of fluid." However, he seems to find it necessary to advance a new theory of the origin of glaucoma, in which he considers the vitreous and not the aqueous as the essential factor. He believes that a change in the volume of the vitreous is the main factor in producing variations in intraocular pressure, and that this change in volume is dependent on alterations in the blood plasma, perhaps especially of its hydrogen-ion concentration.

Certainly these new views on the nature and origin of the intraocular fluids and on the variations in them produced by disturbances in the chemical and physical properties of the blood plasma offer an extremely interesting and important field for progress in both the clinical and experimental study of the etiology and treatment of diseases of the eyes.

Dr. Arthur M. Yudkin, New Haven, Conn., (closing): I want to thank Dr. Lamb and Dr. Wagener for their kind and courteous discussion of this paper. I believe a further study of this problem will reveal more of the physical and chemical signs of cataract formation, and incidentally open a field of investigation of other pathologic ocular conditions.

THE OPTICAL CORRECTION OF CONICAL CORNEA WITH THE CONTACT GLASS.

DONALD H. O'ROURKE, M.D.

DENVER, COLO.

The first departure from the ordinary spherocylindric combinations in the optical correction of conical cornea was advocated by Rählman[2] in 1879, who used lenses of a hyperbolic curve. And it should be noted that Krämer,[2] as recently as 1924, reported ten cases in which a very satisfactory improvement in vision was effected by the use of these lenses. Krämer, however, stated in the same paper that perhaps the best optical effect was obtained with the hydrodiascope of Lohnstein,[3] and that the contact glass was but very little inferior. With the hyperbolic lens the field of vision is very narrow, and only when the apex of the lens is aligned with the apex of the corneal cone, is it of value. Obviously, this alignment obtains in only one position of the eye, therefore their usefulness is limited. The hydrodiascope, altho optically efficient, is difficult of adjustment, and the appearance of the apparatus is such that its use would be confined to the private life of the patient.

The contact glass for the optical correction of keratoconus was devised by Fick[4] in 1888 and improved by Sulzer[5] a year or two later. The glass of these early investigators was properly called a contact glass, in the strict sense of the term, because the thin shell of clear glass was placed on the corneal surface with only a thin film of fluid intervening. In its peripheral portion this glass rested on the peripheral portion of the cornea. This feature presented a serious obstacle to Fick in the furtherance of his work, because his patients were unable to withstand for more than a few minutes the intense irritation of the cornea. As A. Siegrist[6] explained some years later, the two pioneer workers in this field, Fick and Sulzer, were more interested in experimentation for visual acuity with the contact glass, than they were with its clinical applicability.

The first contact glasses which were subjected to prolonged clinical use and which aroused more widespread theoretic and

clinical investigation, were made by Mueller[7] of Wiesbaden. The Mueller device was made of blown glass. It consisted of a thin shell resembling an ordinary prothesis. This was an improvement over the Fick glass, in that a flange was added which was designed to fit the sclera. This flange formed a base, which supported the corneal segment and raised it from direct contact with the cornea. This improvement enabled the glass to be worn comfortably. It was soon learned, however, that no two corneal segments of the Mueller glass were mathematically accurate and, moreover, were often decidedly imperfect, showing high astigmatic errors. It was found to be technically impossible to blow a perfect spherical surface with accuracy. Because of these imperfections, a suitable glass in a given case was obtained empirically, simply by the trying on of a large number of glasses. This was an arduous task for the physician and a trying one for the patient, only to be repeated in case a replacement was required. Attempts to grind the blow glasses to a uniform curvature were unsuccessful. In one such endeavor, 200 shells were broken one after another in an attempt at grinding. Nevertheless, in spite of the obstacles encountered, a number of very gratifying results in improvement of visual acuity were obtained by the aid of the Mueller blown contact shells. (Lauber,[8] Qurin,[9] Scheffels,[10] Siegrist,[11] G. Weill,[12] Clausen,[13] Stock.[14])

In 1920, at Heidelberg, Stock[15] first showed the ground contact glasses as made by the Zeiss firm of Jena. This shell had the scleral and corneal contour of the anterior two-fifths of the globe. The corneal segment, bulging forward, formed a corneal chamber. The corneal segments of the glass were made in three diameters, 13 mm., 12.5 mm., and 12 mm. The height of the corneal segments were 4 mm., 4.5 mm., and 3 mm. respectively. The shell was very thin and light, and more nearly approached the ideal specifications for a suitable contact glass than any made up to that time. Contact glasses of practically these specifications are to be had at the present time. Corneal curvatures of a radius of 6.5 mm., 7.1 mm., 8.1 mm., and 9.0 mm. are available.

B. Von Dohme[16] formulated the following qualifications for an ideal contact glass: First a perfect corneal curvature is essential; second, the availability of a ground-in correction of proper strength; (this point, however, is not an absolute necessity, because a shell without a ground-in correction may be worn with a spherocylindric correction, taken care of by lenses in the usual way); third, the glasses should be graduated according to a defi-

nite scale; next, an accurate fit to the sclera is necessary so that no displacement will occur on movement of the lids; and, lastly, the shell must not be affected by the tears and must not be noticeable to the casual observer. These desiderata, the ground contact glass fulfills very satisfactorily.

There are, however, other considerations in the practical use of the glass which must be taken into account. That a glass must be worn without irritation is paramount. And in this regard, all possible variations and individual tolerances are reported. For example, cases are on record where seamstresses, teachers, technicians and public clerks have worn contact glasses a full working day, month after month, and year after year with no symptoms of distress. On the other hand, it is reported that some patients have tolerated a contact glass in one eye while the fellow eye was absolutely intolerant. The general opinion is, that in a short time a tolerance is established, so that the wearing of the glass may be accomplished for several hours daily. In our experience, however, it is of the first importance to select in the outset a shell of a sufficient corneal height to prevent any possibility of corneal contact. I feel confident that if this precaution is adhered to, any patient with a little determination can wear a contact glass successfully. The final problem to be solved in the successful use of the contact glass is the placement of the shell in position by the patient.

My first difficulty encountered with the case which I wish to report was that of the placement of the shells. It must be understood, that if the desired optical effect is to be obtained, the corneal chamber of the contact glass must be adjusted in position immediately over the cornea, with a sufficient amount of physiologic salt solution between the posterior surface of the glass and the corneal surface of the eye to completely fill the space. Only when this condition obtains, have we substituted for the irregular and strongly refractive conus a regular and much weaker refractive surface. After a few trials in which a one-half per cent solution of holocain in the conjunctival sac preceded the placing of the glass, its adjustment to the eye was accomplished quite readily, but always a certain amount of salt solution was lost in the manipulation, so that air bubbles appeared in the corneal chamber. It was necessary to get rid of these by a rather awkward procedure of introducing salt solution into the chamber between the edge of the glass and the adjacent sclera. Again, it was necessary for the patient to come to Den-

ver from a nearby town in order to have the contact glasses applied. These were undesirable features, if the glasses were to be used by the patient with any degree of satisfaction. Consequently, a simple apparatus was devised, which has proved very satisfactory for placing the contact glass in position. This is done by the patient unassisted, accurately, quickly and with the elimination of air bubbles. The apparatus is shown in Figure 1.

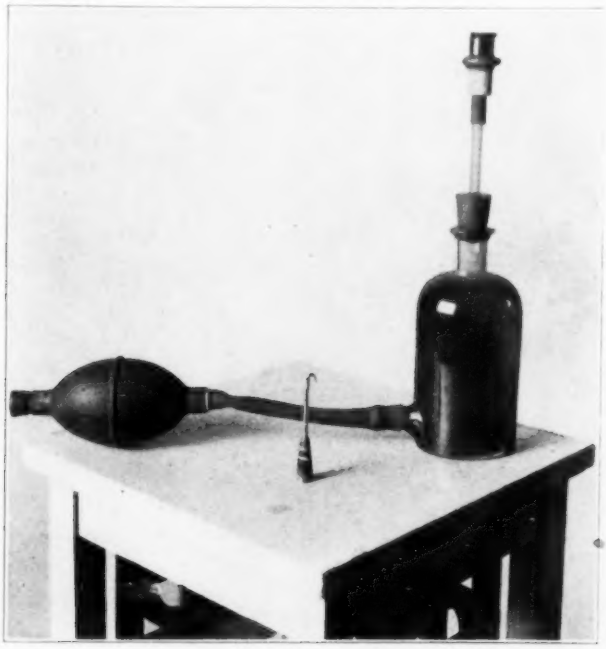

Fig. 1.

To the outflow neck of a small wash bottle is attached an ordinary hand bulb. The wash bottle is filled with water up to the lower margin of the neck, thus leaving a small air space. A piece of glass tubing six inches long and of small diameter is fitted at one end with a rubber cork. The opposite end is fitted with a piece of rubber gas tube an inch and a half long. The gas tube is of a diameter of 15 mm. The free end of this tube is cupped out so that it may receive the convex surface of the contact glass. Over the free end of the tube is stretched a gum rubber finger cot. A small pin point perforation is made in the finger cot at its center. The rubber cork is inserted into the bot-

tle. An application of pressure on the bulb forces air thru the pin hole in the cot. The shell is now moistened and placed in contact with the cot. A release of pressure on the bulb causes the shell to be held firmly in position by air pressure. The concavity of the glass is filled with normal salt solution. The patient retracts his lids as shown in Figure 2, places his eye directly over the center of the shell, the sclera in contact with the flange.

Fig. 2.

He then permits the retracted lids to slip over the margins of the glass. Pressure is now applied with the hand bulb and the contact glass is in place. The removal of the glass is a simple maneuver. Any small blunt instrument, as shown in Figure 1, may be inserted gently between the sclera and the glass, the lids retracted and the shell drawn forward and out.

The case which prompted my interest in this subject was first seen by Dr. Edward Jackson in 1910. At that time the patient was 19 years of age. He complained of poor vision in both eyes, which began five years previously. The vision O. D. was 4/60, and O. S. 2/150. With a—15.00 sph. ⌒ —8.00 cyl. ax. 20° the

vision O. D. was 4/30, and with a —10.00 sph. ⌒ —5.00 cyl. ax. 20° the vision O. S. was 4/12.

In September, 1914, the correction was changed to O. D. —3.00 sph. ⌒ —6.00 cyl. ax. 25°, V = 5/15, and O. S. —6.00 sph. ⌒ —1.00 cyl. ax. 180°, V. = 5/15. These notations were recorded with a 2 to 3 mm. pupil, with the eyes under a solution of pilocarpin gr. 3 to dr. 4, drops 2 instilled three times daily. The correction given in 1914 was worn about six months and not until 1923 were glasses worn again, at which time a 3 centrad prism base up O. D. and a 2 centrad prism base down O. S. were prescribed. These prisms alleviated an annoying horizontal aberration. The vision O. D. with a 1.5 mm. pupil and the prismatic correction was 6/24 and O. S. 6/18. In April, 1927, when the patient first came under my observation, the vision O. D. was 6/30 and O. S. 2/60. This vision was with the prismatic correction only, which he had worn constantly since 1923. Contact glasses with a radius of curvature of 7.1 mm. were applied to both eyes. The vision was improved to 6/12 and 6/20 respectively. Since this time the contact glasses have been worn daily. The patient reads ordinary print comfortably. He conducts his high school classes in mathematics with a great deal more satisfaction and, for the first time in years, recognizes faces distinctly. In short his possibilities in life have been markedly improved.

A consideration of the visual notations given above give some slight concept of the extreme variation in visual acuity resulting from conical cornea. An inadequate conception, however, is conveyed as to the actual effect upon the general visual impressions of my patient affected by the application of the contact glasses. In an effort to make his impression known to you, I quote from him directly; "The contact glasses sweep away nearly all of the harrassing blur that has been before me for years. The thrill of seeing clearly objects for so long seen only indistinctly; the clear cut line of type; the distant horizon; the intervening valley in all its beautiful detail, is something that I never expected to experience again in this life."

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DISCUSSION.

DR. E. E. BLAAUW, Buffalo, N. Y.: Keratoconus is a self limited disease. This means that its clinical course at a certain time comes to a stop. The great question is, when?

In the care of a case of keratoconus, we must first investigate the patient's general condition, with especial reference to the endocrine system. Undoubtedly, the Abderhalden investigations have demonstrated that one or more endocrine glands can be involved. This has suggested, in not too advanced cases, treatment with thymus gland, one tablet every other day, with the use of which Segrist, of Berne, observed improvement, or with thymus-opton, which was very effective in the hands of Schnandizel, of Frankfort.

When, however, the vision is reduced to an extent that glasses do not give an appreciable correction, we should try the contact glass of Zeiss.

To the successful case of Dr. O'Rourke, I can add another. In 1925, a twenty-year old student in Ann Arbor University came with a double keratoconus, wearing heavy myopic lenses prescribed by an optometrist. He counted fingers with O. D. at some two to three meters, while O. S. vision was 2/60. O. D. showed the deep fine lines in the cornea, beautifully demonstrated with the slit lamp, but not in O. S. The vision O. S. could be improved with a strong positive cylinder, axis horizontal. With the appropriate contact glass, the vision O. D. was improved to 6/9. The patient went back to school, wore his contact glass eight to ten hours daily, and finished his fourth and last year without discomfort. I have seen the patient the two following years, and he has broken only one glass. In prescribing this glass, the mentality of the patient must be considered. Another and much older patient, who also showed improvement with a contact glass, did not care to try to use it.

I am surprised that Dr. O'Rourke's patient has had such difficulty in inserting the glass. His case in this respect is unique. My patient was succesful after a few trials, and he did not even need a special contrivance for removing it.

In closing, I want to make von Hippel's statement mine: "The treatment with this prothesis should become in the future the procedure of selection. I would at least, not attack any case of keratoconus operatively before the prothesis treatment has shown itself unsuccessful."

DR. EDWARD JACKSON, Denver, Colo.: The contact glasses for keratoconus were first proposed from a theoretic standpoint, and from that standpoint the whole question was very simple, so that it required

scarcely any study. To get a regular, smooth surface which was continuous with the cornea, thru a fluid with practically the same index of refraction as the cornea—that was the problem. But at least two practical difficulties greatly delay the appreciation of the treatment. One of these is shown by later experience to have been the intolerance of the cornea to the contact glass, and that was removed by making the glass come in contact with the conjunctiva and not touch the cornea. Later observations have seemed to emphasize that distinction. When the glass rests outside of the cornea, the latter is not touched, and when the proper fluid is introduced, as Dr. O'Rourke has shown very distinctly, the glass can be worn all day with no bad effect. In an effort to avoid irritation of the cornea in the earlier trials (I made some of these when the contact glass was first introduced), a local anesthetic was resorted to, but that probably never could be satisfactory. The solution of the problem as to fluid, is to get one that is most agreeable, not irritating, but most nearly physiologic to the cornea. This is probably physiologic salt solution, or some other solution—a mixture of water with a small percentage of sodium chlorid makes a nonirritating solution for most eyes. But this is still to be investigated, because the experience to this time seems to show, that while some corneas tolerate contact with ordinary solutions, there are others that have some particular form of irritability.

The final practical difficulty to be overcome was enabling the patient to place the contact glass, and I think this, with the assistance of the patient, Dr. O'Rourke has very well solved, by the simple method and apparatus that has been in use for a good many months. With that he seems to have no serious difficulty. But Dr. O'Rourke has also been particularly fortunate in getting a patient who went ahead and got a rather unusual intellectual development with such a high conical cornea and such poor vision, without being subjected to surgical procedure. The final success of the case may rest on this, and this comes in with the point that Dr. Blaauw has raised with reference to the endocrines. His prescription for contact glass treatment was certainly helpful, but it was perhaps essentially helped by repeated thyroidectomies. In this case, it was a partial thyroidectomy; in another case, where there was a long history of very serious impairment of alimentary function, a similar result in change of nutrition was brought about, not by thyroidectomy, but by gastroenterostomy.

These cases, altho they are rather rare, are not much more rare than gastric cases that yield brilliant success after operation. These cases require careful study. They are not to be solved by leaving them, as most of them have been left heretofore, as my cases have been left, to the best sphero-cylindrical correction that can be worn. The contact glass now is not something theoretic, but a practical procedure to apply to all cases of very high conical cornea.

Dr. Robert Von der Heydt, Chicago: During the last two years, I have had available to me six cases of keratoconus. I will mention the interesting points about each one.

Case 1. E. C., female, age 45. Because of 20/30 vision in the one eye, a contact glass is not needed.

Case 2. F. B., male, age 21. 13 mm. megalocornea. Vision 20/100,

with contact glass 20/40. The glass was ordered and paid for by the patient. After three months, when it arrived, he reported that he was not interested because he is being cured by an osteopath.

CASE 3. A. J., female, age 23. Vision 10/200, with contact glass 20/40.

CASE 4. J. B., female, age 37. Keratoconus with high myopia. She refused to try the contact glass, her relatives said it was not necessary for her to see better as she just washed dishes.

CASE 5. Mrs. A., now age 40. A patient I had seen twelve years ago. Vision in the right eye, fingers at three feet; left eye, fingers at two and one-half feet. This is the most advanced case, with marked corneal clouding. Vision with contact glass 20/60 in each eye. The same contact glass usually gives identical results in either eye. The lowering of vision due to optical distortion in keratoconus is entirely eliminated. That due to the usually slight superficial scar formation remains. Hence the astounding betterment in visual acuity.

CASE 6. F. K., male, age 27. This is the patient who is on exhibition here today. Vision 4/200, with glass 20/30 plus. He inserts glass under normal salt water. There is a slight congestion of the conjunctiva today owing to the fact that he inserted the glass using ordinary tap water. He wears it for eight or ten hours at a time without discomfort.

Because of the fact that it is difficult, in trying these glasses, to find out which glass is the proper one to use, there being four different contact glasses of varying heights in the trial set, it was suggested to me by the representative of Carl Zeiss, that some means might be devised whereby the elevation of the cone could be measured. With that in view, I constructed a little silver instrument on the order of a tonometer to measure the conus. With it you can gauge the height of the elevation of the conus above normal. The necesity of trying all four of the contact glasses is thus avoided.

DR. D. H. O'ROURKE, Denver, colo., (closing): I wish to thank Dr. Jackson, Dr. Blaauw and Dr Von der Heydt for their discussion of my paper.

Dr. Blaauw in his discussion mentioned a special device which was used to remove the contact glass. The removal of the glass is a very simple matter. Any blunt instrument may be inserted between the glass and the sclera, the lids retracted with the thumb and forefinger of the free hand, and with gentle traction, the contact glass is removed.

The introduction of the contact glass however, without the appearance of air bubbles in the chamber, I found quite difficult. But by the use of the device which I have demonstrated here and have passed among you, the placing of the contact glass is made very easy and is accomplished by the patient without assistance.

I should like to call your attention to, and have you examine, a patient in the back of this assembly room. He has been wearing a contact glass for the past seven hours. He places his glass in position by immersing his face in a bowl of water.

Of the various fluids I have used in the contact glass, normal salt solution is to be preferred. Ringer's solution, two per cent dextrose solution and glycerin, sixteen drops to the ounce of water, have been tried and discarded.

There is a curious phenomenon which my patient noticed during our first experiences with the contact glass. He observed, that if he wore the contact glasses for fifteen minutes or so and then removed them, he could see to read very much better. Just what caused this improvement in his vision at the near point I am unable to say. After a few months, however, this phenomenon no longer occurred.

Let me caution you concerning the cloudiness of the fluid which occurs in the corneal chamber of the contact glass. Sometimes, this is noticeable within half an hour, and again, several hours may pass without clouding of the fluid. This point is worth remembering. Nothing has gone wrong, but the glass must be removed and the fluid changed.

CLINICAL STUDIES IN SLITLAMP OPHTHALMOSCOPY.*

1. Cystic Degeneration of the Macula.
2. Serous Transudates Near the Optic Disc.
3. Subretinal Exudates and Detachment of the Retina.

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The slitlamp magnifying hand ophthalmoscope with which the following observations were made has been described elsewhere.[1,2] It possesses two characteristics which are of importance in relation to the present study: (a) the illuminating beam which casts the image of a narrow slit upon the retina can be directed obliquely to the axis of observation, so as to provide oblique focal illumination of fundal details analogous to that obtainable in the anterior segment of the globe with the slitlamp corneal microscope; (b) the definition of the ophthalmoscopic image is heightened by the suppression of its chromatic aberration either thru the introduction of appropriate color filters into the illuminating system or thru the use of an achromatizing lens in the observing system; and the image so sharpened may be magnified with the aid of a small telescopic ocular. Thru these means, certain ophthalmoscopic details may be seen which are not recognizable with other instruments.

1. Cystic Degeneration of the Macula.

The group of cases which are here to be reported present the appearance, on ophthalmoscopy with the ordinary type of instrument, of a hole in the macula, but with the slitlamp ophthalmoscope the cystic nature of the defect is readily recognized, as is seen in the accompanying illustration. (Fig. 1). The cases

*From the Wilmer Ophthalmological Institute of the Johns Hopkins Medical School and Hospital.

which have hitherto been designated under the general rubric of hole in the macula fall into three groups: (1) those which follow injury, (2) those which follow inflammatory diseases of the eye, and (3) those which occur spontaneously without antecedent ocular diseases. It is to this third group that the cases which are to be described belong. That a true hole in the macula can occur, has been amply demonstrated by anatomic studies.[3] There is, furthermore, histologic evidence that cystic degenera-

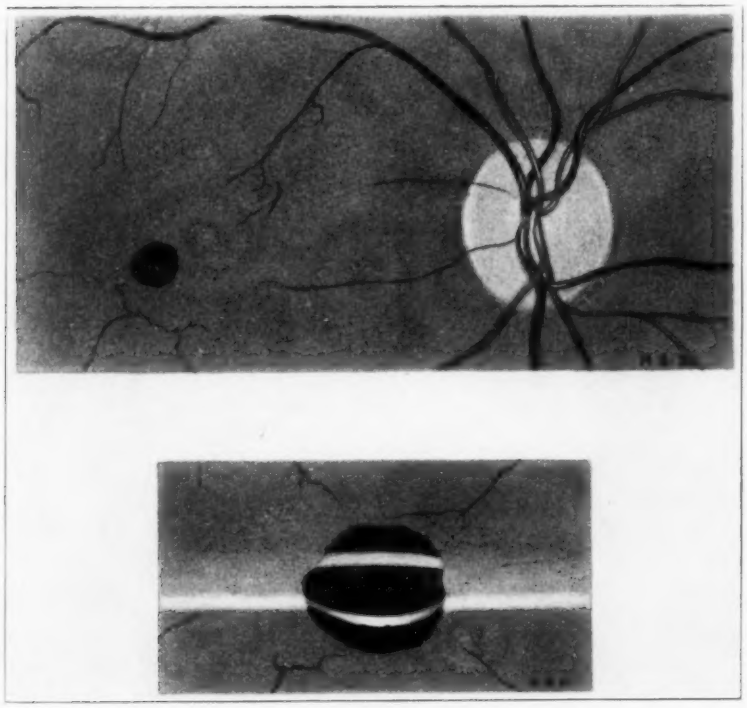

Fig. 1. Senile cystic degeneration of the macula.

Above.—Appearance with ordinary ophthalmoscope suggestive of hole in macula.

Below.—Appearance with slit lamp ophthalmoscope demonstrating cystic character.

tion of the macula can occur, and it has been suggested that the cases of spontaneous hole in the macula may be the result of the rupture of a macular cyst.[4, 5] The histologic studies indicate quite clearly that the cysts develop as a result of a hemorrhagic or serous exudation. There seems to be no fundamental anatomic difference between those which occur

as a complication of prolonged intraocular inflammation (iridocyclitis) and those which occur spontaneously in elderly people, where no doubt an arteriosclerotic affection of the macular vessels is responsible for the original serous or hemorrhagic exudation. Most of the cases of spontaneous hole in the macula described in the literature have been bilateral, and almost all have occurred in elderly women.

Report of Cases.

Case No. 1. (Miss W.) White female, aged 60. Past history: The patient had pneumonia last year, in the course of which a rather severe diabetes developed. After the infection

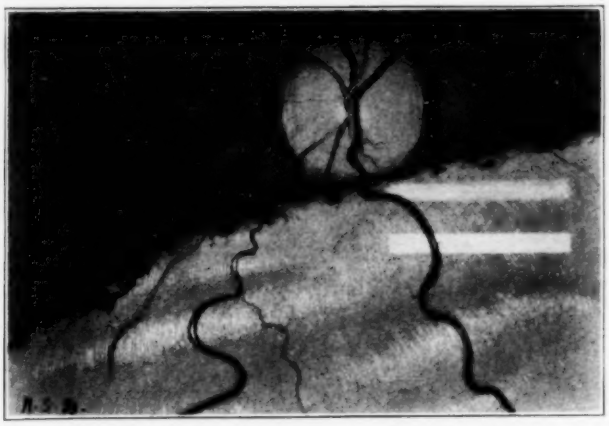

Fig. 2. Detachment of the retina 2 years after onset. Note the subretinal fluid is optically clear.

passed off, the diabetes became mild and was readily controlled by diet. Present illness: Complains of a black spot before her eyes for some weeks. The left eye "blurs" at times. Examination: Right eye—vision 6/12. Media clear. No signs of retinal arteriosclerosis noted. In the right fovea, there is a small, dark red, sharply circumscribed spot about 1/3 the diameter of the disc in size, which with the ordinary ophthalmoscope appears to be a hole in the macula. Its appearance as seen with the slit-lamp ophthalmoscope is shown in the accompanying figure. Left eye—vision 6/9, 6/6 almost. Ophthalmoscopic examination negative. General examination by Dr. Eugene Leopold: Blood pressure normal; mild diabetes under satisfactory control;

chronic gastric hyperacidity; mild chronic arthritis. Course: No change in vision or in eyegrounds in three months.

Case No. 2. (Mrs. N.) White female, aged 50. Past history not relevant. Present illness: Has noted failing vision for the last eight months, more rapidly so for the last four weeks. Examination: Right eye—vision 6/60. Media clear The larger retinal arteries are of good caliber but show marked beading and slight increased visibility of their walls. There is marked arteriovenous compression. At the upper temporal margin of the disc, a small bed of dilated capillaries and one flame shaped hemorrhage are seen. The retina in the macular region has a glassy, watery appearance, and fine web like irregularities in its structure are seen on indirect illumination. There are many colloidal excrescences of Bruch's membrane. In the fovea there is a dark red, horizontally oval, sharply outlined spot about 2/3 the size of the disc, which with the slitlamp ophthalmoscope appears definitely to be cystic. Left eye—vision 6/24. There are a few spicules in the lens. Retinal blood vessels show much the same condition as in the other eye. There are many petechiae in the macular region, but no foveal lesion.

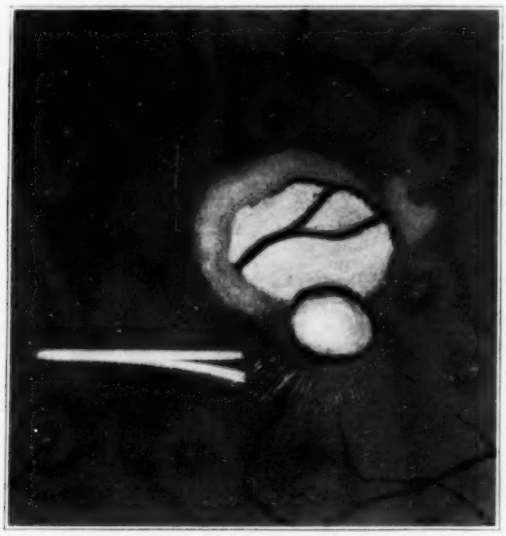

Fig. 3. Boy aged 10 years with old choroidal scar at the macula. Round fibrous nodule at the margin of the scar causing localized tent like detachment of retina. Slit lamp ophthalmoscope shows extent of the detachment and also the clarity of the subretinal fluid.

General physical examination by Dr. Frank R. Smith: Blood pressure 205/100. Heart not enlarged. Urine shows a trace of albumin but no casts. Examination otherwise negative. Course: Vision has fluctuated irregularly for over a year. At last examination, the cyst can no longer be clearly recognized and there is beginning disciform degeneration (Scheibenformige Entartung) in each macula.

Case No. 3: (Mrs. P.) White female, aged 58. Past history: Negative. Present illness: Blurred vision of the right eye for six months. The patient notices a small, dark circle in the center of her field of vision when she reads. Examination: Right eye—vision 6/12. Media clear. No retinal arteriosclerosis noted. In the fovea, a small cyst identical in appearance with that seen in Case No. 1. Left eye—vision 6/6. Ophthalmoscopic examination negative. Course: Six months later the patient returned saying that she now noticed a similar dark ring before her left eye. Examination at this time: Right eye—vision 6/12. The foveal cyst has disappeared entirely and only a slight irregularity in the choroidal pigmentation beneath the macula remains. Left eye—vision 6/9. There is a typical foveal cyst similar to that previously seen in the right eye. General physical examination: completely negative. Urine negative.

Discussion.

The cystic nature of the macular changes in the cases reported is so clearly demonstrable with the slitlamp ophthalmoscope that additional proof need hardly be given. The disappearance of the cyst in the right eye of case No. 3 may, however, be mentioned in support of the diagnosis, for it is inconceivable that a hole in the macula should disappear in so complete a manner. In case No. 3, no ophthalmoscopic or clinical evidence of arteriorsclerosis was found, but the patient is 58 years old and no other more likely explanation for the foveal lesion presents itself.

2. Serous Transudates Near the Optic Disc.

A problem of major importance both to the clinical ophthalmologist and to the ophthalmic pathologist is the mutual reconciliation and reinterpretation of ophthalmoscopic and histologic pictures. In many respects this is not an easy task. The retina is a highly translucent tissue, and slight local changes in

the index of refraction of its tissue fluids may reveal themselves to the ophthalmoscopist as gray or whitish patches, tho microscopic sections of the same region may show no abnormality. On the other hand, lesions which are quite obvious to the histologist may largely escape ophthalmoscopic detection if the color and translucency of the lesion differ little from that of the surrounding retina. Among this latter group are serous extravasations in the retina, which occur so commonly in albuminuric

Fig. 4. Flat detachment of the retina in the macula in albuminuric retinitis. Note turbidity of the subretinal fluid. This picture is to be distinguished from retinal edema by the absence of pitting at the fovea.

retinitis, choked disc, etc. There are, in general, two forms of such extravasations. Those which occur in the nerve fiber layer of the retina spread out flatwise in this layer, but do not, unless they are very large, extend deeper into the retina. Like the flame shaped hemorrhages which occur in the same region, their outlines are ill defined. On ophthalmoscopic examination they appear, if at all, as faint grayish "cotton-wool" spots. The second form of serous extravasations embraces those which occur in the deeper layers of the retina. Here, as is well known, the accumulation of fluid usually takes place between the fibers of the outer fibrillar layer, pushing the fibers aside so as to form irregular or oval spaces.

In the macular region these serous accumulations usually are found in the layer of Henle. They are often fairly large, almost always oval, and tend to string out in a radial spoke like arrangement about the fovea. When they become infiltrated with hyalin or lipoid material, they are readily visible with the ophthalmoscope and constitute the so called star shaped figure at the macula. In the region about the optic disc, these serous accumu-

lations are less often round or oval, and tend to fill an irregular network of interlacing channels. No doubt this difference is the result of slight local differences in the anatomy of the Müller supporting fibers of the retina. This network, which is a well recognized histologic structure, is of such size as to be well within the limits of ophthalmoscopic visibility, but the difference in translucency of the serous accumulations and of the surrounding retina is so slight as to escape notice. With the increase of definition brought about by the suppression of the chromatic aberration of the ophthalmoscopic image as attained in the new instrument, this network can be seen in most cases of albuminuric retinitis and of choked disc, (Fig. 5).

Fig. 5. Serous exudate in the retina on the nasal side of the optic disc in a case of albuminuric retinitis.

3. Subretinal Exudates and Detachment of the Retina.

The use of the very narrow, obliquely focused ophthalmoscopic beam in the study of cases of detachment of the retina has rendered possible some observations on the character of the subretinal fluid. In cases of long standing detachment this fluid usually appears entirely clear. The beam cannot be seen from the place where it leaves the retina to that where it strikes the choroidal lamella—the intervening space is optically as empty as the normal aqueous (Figs. 2, 3). In very fresh detachments of the retina, and especially in the flat detachment so commonly seen in albuminuric retinitis, the subretinal fluid is relatively turbid and the passage of the beam from the retina to the choroid is clearly outlined, (Fig. 4).

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DISCUSSION.

Dr. Robert Von der Heydt, Chicago, Ill.: I have not had experience with the slit ophthalmoscope designed by Dr. Friedenwald, because it is not available. It is a very important and extremely valuable instrument, and its applicability must be to allow differentiation between retinal separation and separation overlying a tumor.

For refinement in fundus diagnosis, I use the Gullstrand binocular ophthalmoscope as well as the ordinary one with complete red free illumination, derived from a microarc lamp.

I am especially interested in the clinical diagnosis of macular degeneration, as exemplified by cyst and hole formation. These occurrences are not limited by age or sex. Their frequency has been brought out by the examination of recruits in the late war. A common etiology is trauma. Heredity and their occurrence secondary to neuroretinitis are next in frequency.

Stereophotography of the fundus has shown, in some cases, that the cyst still existed when a hole in the macula had been suspected. This is because of the visibility of a reflection from the thin anterior convex cyst wall seen by the camera. In another case, a double set of radial retinal folds have shown an elevated lesion to be just adjacent to and not of the macula.

Another aid in the diagnosis of the presence of an anterior cyst wall is the behavior of its reflex when seen in red free light. On lateral motion of the ophthalmoscope (not a rotation of the mirror) the anterior cyst wall reflex moves in a with direction. If the wall is broken and a hole exists, the reflex has an against motion, presuming the hole has a glossy lining capable of producing a reflection.

Red free light also discloses macular pigment. If enough has formed that an area appears black with ordinary light, red free light will show it yellow. Fresh pigment of recent formation is lemon yellow in color. In ordinary light, it may be quite red and thus simulate a minute macular hemorrhage.

Dr. Friedenwald is to be congratulated on presenting a new method of fundus observation, the application of a slit image in the diagnosis of changes in the contour of the retinal surface.

Dr. Harry S. Gradle, Chicago, Ill.: Dr. Friedenwald's paper is a beautiful illustration of the value of observing the fundus with various types of illumination. The focal illumination of the fundus, of course, antedates this instrument. The original method proposed by Koeppe of slit lamp observation of the fundus is extremely complicated. In this method,

the fundus picture is brought forward by means of contact glasses upon the anterior surface of the cornea, until the fundus picture lies 15 millimeters beyond the cornea. With this apparatus, the fundus can be observed with a binocular microscope. However, this method of observation requires conscientious, painstaking effort on the part of the patient. When things work well, I believe a great deal of value can be gained by studying the pathologic lesions of the fundus by slitlamp microscopy, but only in the exceptional case are we able to use it, and then only for study. It is not adapted for routine work at all. I believe Dr. Friedenwald's instrument will obviate that difficulty.

There is still another way we have tried that is satisfactory. The patient is anesthetized lightly with any usual anesthetic, and the Koeppe contact glass is applied over the cornea. That is a little bit difficult until one is experienced. The method of application is like that of the contact glass that Dr. O'Rourke spoke of. Then, the fundus is observed with the hand slit lamp made by an American firm. The important thing is the focal illumination of the retina that we gain. It is not a routine office measure. It is applicable only in certain cases, where differential diagnosis is required. However, it is extremely simple to use after one obtains the knack of applying the contact glass, and I recommend it to your attention.

Dr. Jonas S. Friedenwald, Baltimore, Md., (closing): In regard to Dr. Von der Heydt's statement concerning the occurrence of macular degeneration, there are several clinical types in which this is found. There are the traumatic cases, which occur at all ages; there are the cases seen in relation to chronic iridocyclitis at all ages and in both sexes, and finally there are the arteriosclerotic and senile cases, which are said to occur more frequently in elderly women. My own experience has been that of the four cases of the latter group, which I have seen in the last year and a half, and three have been in elderly women.

The question of the other methods of slit lamp illumination of the fundus which Dr. Gradle raises is very interesting. The first slit lamp arrangement for examining the fundus antedates Koeppe by some fifteen years, and was produced by Hugo Wolff of Berlin. It was a very complicated instrument, altho the general plan of the illuminating system is not very far from that which I have used, and I do not claim any originality for this aspect of my instrument. Koeppe developed his method eight or ten years ago for observation of the fundus by the use of the corneal contact glass and slit lamp microscope. In addition to the great practical difficulties in using the contact glass, which Dr. Gradle mentioned, there is this theoretic difficulty, that the imposition of the contact glass upon the eye enormously increases the spherical aberration of the optical system, and thus renders the definition of the ophthalmoscopic picture very imperfect. It seems to me this is the reason why, in spite of the fact that the Koeppe instrument has been in use for eight or ten years, no significant discoveries have been reported by its users. I have had no experience with the ingenious use of the hand slit lamp and corneal contact glass which Dr. Gradle describes.

SUTURE FILTRATION OPERATION FOR GLAUCOMA SIMPLEX.*

GRADY E. CLAY, B.S., M.D.

ATLANTA, GA.

Ophthalmologists with few exceptions have adopted some type of filtration operation for glaucoma simplex. The Elliot Trephine is the one method most generally used of the more than fifty such operations, but with this procedure there are many serious objections, such as late infections, late iritis and hypotension.

The suture filtration operation which is to be presented, apparently meets these objections, in that it gives a small avenue of escape of aqueous and a broad area of filtration. The operation was first done one and half years ago, and the case was followed for four months with good results. Since March of this year, eleven other cases have been treated with such excellent results, that the operation is herewith presented without further experience, with the hope that other surgeons will try this method and report their experiences.

The preparation of the patient is the same as in other propedures, and miotics are omitted on the second day before the operation. The anesthesia used is the instillation of 4% cocain, and the injection of a few mm. 1% novocain and adrenalin under the conjunctiva over the insertion of the internal and external recti. The first step of the operation is the same as in the Elliot trephine. A thick conjunctival flap is dissected down to the limbus (Fig. 1).

The second step is done with a broad keratome. The eye is fixed in the usual manner, while the assistant holds the conjunctival flap down. The point of the keratoma passes into the sclera 3 mm. back of the limbus, and as it passes into the anterior chamber, the assistant pulls the flap up over the keratome so that operator may follow the movement of the keratome. A broad incision should be made; with the flap held up by the assistant, the operator does a broad iridectomy (Fig. 2).

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Fig. 1

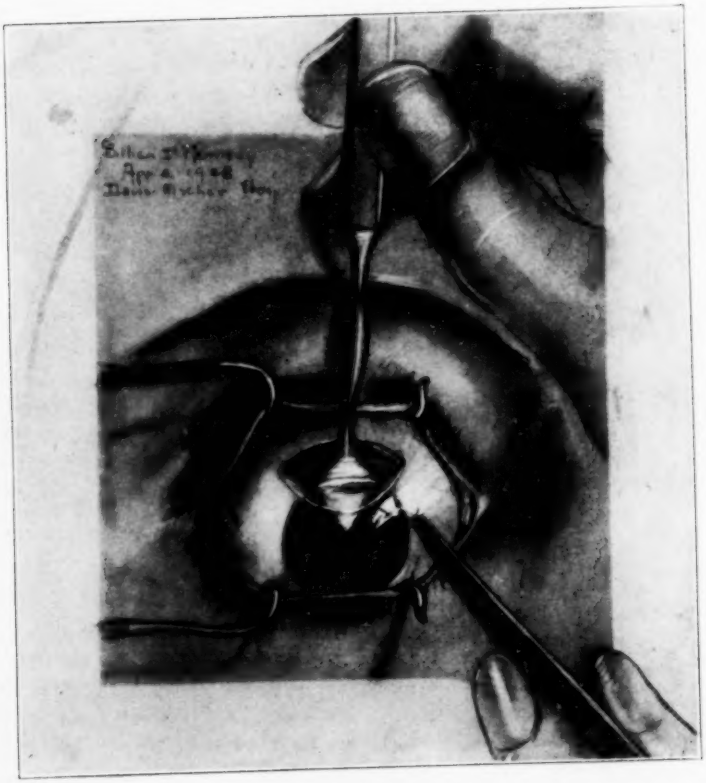

Fig. 2

The most important step in the operation is the proper placement of the sutures. The assistant pulls the conjunctival flap well down. The operator, with a pair of iris forceps, lifts up the sclerocorneal lip, and with a mattress suture, using a fine cutting curved needle and No. 1 black silk, passes it thru into the anterior chamber, and then it is brought out thru the incision; the second needle is passed in a similar manner, 1 mm. to the side (Fig. 3).

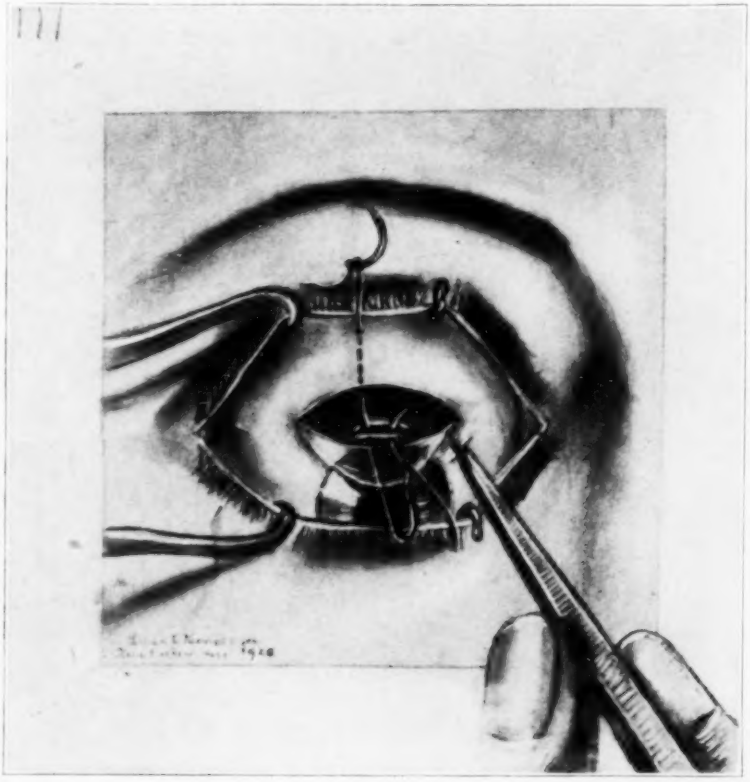

Fig. 3

The conjunctiva above is separated from Tenon's capsule by blunt dissection, and then the sutures are passed up under the conjunctiva for a distance of 4 mm. and brought out and loosely tied. Then the conjunctival incision is closed with a continuous or four interrupted sutures (Fig. 4).

Atropin is instilled, a pad put on the operated eye, the patient

is put to bed for three days, and then discharged from the hospital to return for daily treatment. The eye is dressed daily, using boric acid irrigation and the instillation of atropin and argyrol. The small conjunctival sutures are removed on the fourth day, and atropin discontinued on the sixth day. On the tenth day, the drainage suture is removed; the eye is cocain-

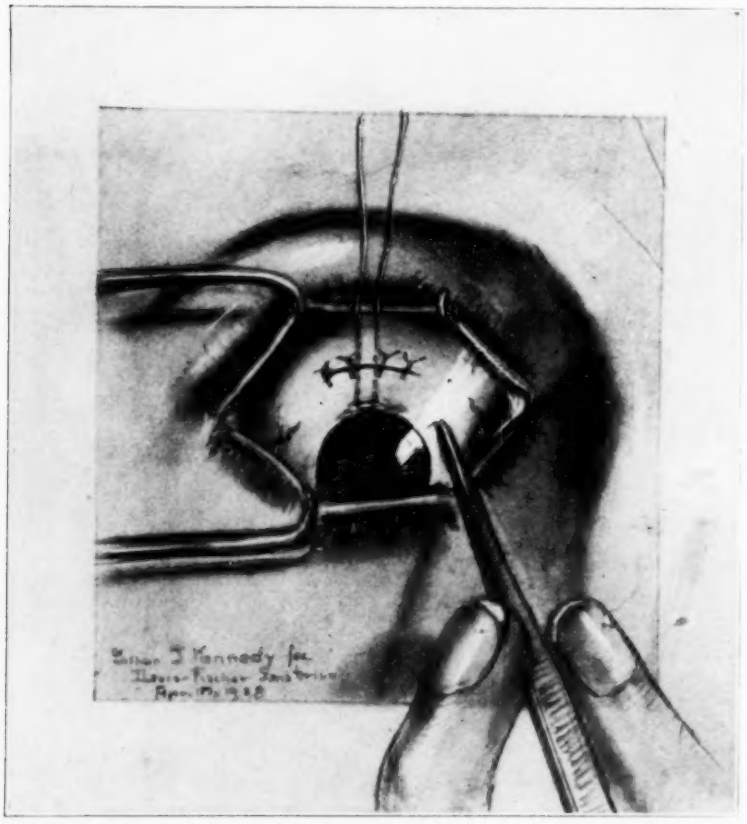

Fig. 4

ized and well cleansed; a speculum is placed and the area around the sutures cleansed with alcohol; the suture is pulled up, one thread cut, and the suture is then removed with ease.

Report of Cases.

Case 1. Mrs. D. C. Age 44. Admitted to Emory University eye clinic March 1, 1928, complaining of gradual loss

of vision. Vision R.20/70; L.20/50. Tension R., 60 mm.; L., 45 mm. (Schiötz). Diagnosis: Glaucoma Simplex.

March 3rd. Patient entered hospital and suture filtration operation done on the right eye without difficulty. The following day, the eye was dressed; very little reaction; anterior chamber formed; pupil well dilated; patient discharged from hospital.

March 19th. Filtration suture removed without difficulty and eye quiet. Tension, R., 15; L., 48.

March 22nd. Patient entered hospital for operation on left eye. Operation done without difficulty. The following day the eye was dressed and found to have a marked conjunctivitis with mucopurulent discharge; anterior chamber partially formed; dressing left off; eye irrigated with boric solution, and 20% argyrol instilled every three hours. On the fourth day chamber fully restored and conjunctivitis much improved; conjunctival sutures removed and patient discharged. Ten days later filtration suture removed and eye quiet.

April 2nd. Tension R., 16; L., 18. April 16th. Tension same. July 2nd. Tension R., 15; L., 15. O. U. shows a large area of filtration above.

Case 2. H. C. Age 53. First seen September 27, 1927. Four months ago began to notice blurring of vision. At that time, right eye nearly blind; since then, sight has become rapidly worse, lately some pain and redness. Vision R., nil; L., light perception. Tension R., 60; L., 50. Diagnosis: Glaucoma Simplex.

March 3, 1928. The suture filtration operation done in usual manner without difficulty; following day marked congestion of conjunctiva especially above; good filtration; anterior chamber not formed.

Fourth day. Marked congestion of conjunctiva; the conjunctival sutures removed; good filtration.

March 10th. Suture filtration operation on the left eye without difficulty. Following day, marked congestion of conjunctiva, some discharge, chamber partially formed.

Fifth day. Eyes fairly quiet; patient discharged from hospital.

Tenth day. Filtration suture removed.

April 5th. Eyes quiet. Tension R., 14; L., 15.

May 21st. Tension R., 13; L., 13. Good filtration extending up to original suture line in fan shape.

July 2nd. Tension R., 15; L.18. O. U. good filtration.

Case 3. Mrs. T. N. Age 53. Admitted to clinic March 27, 1928, complaining of dimness of vision beginning in September, 1927, especially right eye, and has gradually become worse; now blind in right eye. Family history—negative. Vision R., light perception; L., 20/40. Tension R., 34; L. 38. Diagnosis: Glaucoma Simplex.

April 12th. Patient entered hospital for operation on right eye. Operation done in usual manner, a broad iridectomy obtained and filtration sutures placed, and the conjunctival incision sutured; at this point the assistant pulled out the filtration suture by mistake; it was decided to rely on the iridectomy.

Second day. Eye fairly quiet; chamber formed; patient discharged from hospital.

April 19th. Patient entered hospital for operation on left eye. Operation done in usual manner without difficulty. On second day, eye quiet and chamber partially formed; good drainage. On third day, eye fairly quiet; chamber formed; conjunctival sutures removed; patient dismissed from hospital.

May 11th. Eyes quiet. Tension R., 25; L., 15.

June 14th. Vision R., light perception; L., 20/40. Tension R., 28; L., 18.

June 25th. Tension R., 35; L., 20. Eyes quiet.

July 2nd. Tension R., 25; L., 18. O. S. Shows good filtration.

Case 4. Mrs. L. P. Age 40. May 14, 1928. Three years ago, vision began to fail in right eye, which has been blind for past year. One year ago, vision rapidly began to fail in left eye, and now barely able to walk about house. There has been no pain. Vision, R., nil; L., counts fingers at two feet. Tension R., 55; L., 48. Diagnosis: Glaucoma Simplex.

Patient entered hospital May 17th, and suture filtration operation done on right eye. Second day after operation, chamber formed; pupil well dilated; pillars free; good filtration extending up to and above suture line.

May 23rd. Filtration suture removed; eye quiet. Tension R., 14; L., 45.

May 31st. - Patient entered hospital for operation on the left eye, and operation done without difficulty. Second day, marked congestion of ocular conjunctiva above; chamber formed; pupil well dilated; pillars free. Third day. Conjunctival sutures removed; eye fairly quiet; patient discharged from hospital.

June 10th. Eyes quiet; filtration suture left eye removed

June 18th. Eyes quiet; broad area of filtration extending up and fan shaped beyond original suture line. Tension R., 12; L., 12.

July 2nd. Tension R., 15; L., 18. Good filtration.

Case 5. Mrs. M. D. May 10, 1928. Age 70. During the past year, vision has gradually become worse, now almost blind; has had no pain. Vision R., light perception; L., objects 1 foot. Tension O. U., 60.

May 30th. Patient entered hospital for operation on left eye. Suture filtration operation done without difficulty.

June 1st. Marked congestion of conjunctiva; anterior chamber formed; pupil well dilated; pillars free; good filtration.

June 11th. Conjunctiva still congested, only slight discharge; pupil well dilated. Tension normal; patient discharged.

June 18th. Attempt to remove the suture, and after much traction, it pulled away from the sclera lip but broke off and part of suture left buried under the conjunctiva; eye fairly quiet; broad area of filtration. Tension R., 50; L., 16.

July 5th. Eyes quiet. Tension R., 48; L., 15. Good filtration.

Case 6. Mrs. B. F. June 7, 1928. Age 30. Nurse. Blind in right eye since childhood from injury. Vision of left eye getting poor during past year, and has noticed halo around lights. Vision R., Nil; L., 20/30. Tension O. S., 52. Diagnosis: O. D., Phthisis Bulbi; O. S., Glaucoma Simplex. Patient entered hospital on same day and suture filtration operation done on left eye without accident. The third day after operation—eye quiet; broad area of filtration; chamber formed; pupil well dilated; pillars free; conjunctival suture removed and patient discharged.

June 17th. Eye quiet; large area of filtration; filtration suture removed. July 2nd. Tension O. S., 20. Vision, 20/30.

Case 7. Miss H. W. June 3, 1928. Age 44. During the past month, has noticed a blurring of vision. Vision O. D., 20/30; O. S., 20/40. Tension O. D., 35; O. S., 45. Diagnosis: Glaucoma Simplex.

June 7th. Patient entered hospital and suture filtration operation performed on the left eye. Unintentionally a peripheral iridectomy done; otherwise operation in usual manner. Third day after operation, eye fairly quiet; anterior chamber formed; broad area of filtration; conjunctival sutures removed; patient discharged from hospital.

June 17th. Filtration suture removed; eye quiet. Tension

O. D., 25; O. S., 15. July 3rd. Tension O. D., 20; O. S., 16. Using esserin right eye twice daily. Left eye quiet. Very broad area of filtration. Vision R., 20/30; L., 20/30.

The suture filtration operation has been done with the idea of producing a broad area of filtration. The suture produces a small fistulous opening thru the sclera and under the conjunctiva up to the suture line. In addition to this, in every case, the area of filtration seems to definitely broaden out as it passes upwards. In no case has there been the small bleb formation over the filtration opening as is seen after other filtration operations. The small filtration area formed by the suture thru the sclera keeps the tension of the eye at a more normal level, and the long broad area of filtration under the conjunctiva seems to make the development of late infections impossible.

The only disadvantage which might arise is an infection from the conjunctiva extending along the sutures to the interior of the eye. In two of the cases reported, there developed on the second day a severe conjunctivitis, and no complication developed. Since the flow of aqueous is upwards along the suture line, it would seem remote for an infection to travel down along the suture; however, that objection could be overcome by burying the sutures under the conjunctiva and Tenon's capsule.

Summary.

(1). The operation is simple in technic and devoid of danger.

(2). There has been no delay in the restoration of the anterior chamber.

(3). The area of filtration is broad, passing to the limit of the suture line.

(4). The eye usually becomes quiet on the third day and remains so.

(5). The suture is well tolerated by the eye.

(6). No case of hypotension or late iritis in twelve cases.

I wish to thank Dr. Phinizy Calhoun and his associates in the Department of Ophthalmology of Emory University for their generous assistance.

DISCUSSION.

Dr. H. W. Woodruff, Joliet, Ill.: Nearly every operation for glaucoma involves the iris in some way. Many of them, as does the one we are to discuss, include an iridectomy. Elliot advises an iridectomy in his trephining operation, not that the iridectomy in itself is important, but that prolapse of the iris must be avoided. That many glaucomatous eyes are saved by some form of external drainage is true. That many are saved by iridectomy, with no sign of external drainage, is also true. If iridectomy in itself is to be of benefit, it must include the iris root. At the Colorado Springs meeting, three years ago, I presented a paper on deep iridectomy. I attempted to show the impossibility of removing iris root thru an incision made with the keratome, and the possibility of removing iris root thru an incision made with a cataract knife. I am still more strongly convinced, after 6 years of experience with the operation, that it offers the most in chronic simple glaucoma as well as in some other types.

The point which I wish to make is this: Use the suture as Dr. Clay does, if you wish—I see no objection to it, if it is removed in ten days—but make the incision as near the plane of the iris as possible, and follow the technic of Török in removing the iris. Namely, draw the iris downward toward the pupil with the iris forceps held parallel with the wound. This gives the best chance of incising the iris at its ciliary attachment. This may be preceded by a slight cyclodialysis which still further insures root iridectomy. Permanent foreign body drains are not tolerated by the eye. Temporary ones as is the nature of Dr. Clay's may be of advantage. I am inclined to think, however, that the removal of a piece of sclera from the margin of the corneoscleral flap would be just as certain to give permanent external drainage.

Dr. Grady E. Clay, Atlanta, Ga., (closing): I appreciate Dr. Woodruff's discussion.

I have nothing further to say, except that we have done this experimentally on animals, on dogs particularly, and there is shown a definite avenue of filtration. I believe that the smaller the area of filtration, the better results we will get in glaucoma; and in the course of time, I think these small filtration scars will be slight and the epithelium will become permanent.

The same thing can be applied in trephining. The fault of most filtration operations is that there is too small a filtration area. If this suture is inserted at the border of the trephine opening and then carried backward, I believe we will have established in a trephine operation a broad area of filtration likewise.

TRANSACTIONS

OF THE

THIRTY-THIRD ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OTOLARYNGOLOGIC SESSIONS

POSTTONSILLITIS PYEMIA.*

RICHARD WALDAPFEL, M.D.**

VIENNA, AUSTRIA.

Of all the complications that can occur after angina, postanginal pyemia has been the most extensively investigated during the last four years. As a result, certain problems have arisen which still require definite explanation. Fraenkel believes in a primary thrombosis of the tonsillar veins. Exceptionally, a suppuration of a lymph gland lying on the jugular vein may infect the blood stream. Uffenorde holds Fraenkel's exception to be the general rule; he requires histologic investigation of several sections thru the tonsil and the inflamed surrounding tissue in order to clear up the problem.

Zange's standpoint is the same as Fraenkel's, but as a result of the above supposition, he recommends tonsillectomy as a therapeutic procedure, followed by exposure of the internal jugular vein and all of its accessory branches. In case the vein is thrombosed, he recommends ligation with splitting of the ligated portion of the vessel.

These are the most detailed studies along this line during the last few years and contain the bibliography of numerous lesser references, which I need not enumerate here.

The most important points for discussion are:

(1). The original focus (whether primary thrombosis of the tonsillar veins or primary thrombosis of the internal jugular vein).

(2). The therapy, which should be based on an exact knowledge of the underlying pathology.

My investigations by means of serial histologic sections have served to throw light upon these disputed points.

The clinical picture in these cases varies. Usually, tho not invariably, a few days after the beginning of an attack of ton-

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*From a paper accepted for the Copenhagen Congress, 1928, but not read. Read by invitation before the Academy.

**Assistant Klinik Prof. Hajek.

sillitis a chill occurs. There is a slight tenderness over the region of the sternocleidomastoid. The patient looks septic. A pleuritis often occurs. There is usually fatal termination. Autopsy shows in many cases a thrombosis of the internal jugular vein on the affected side. Practically always, a parapharyngeal abscess is found, which point will be spoken of later.

* * * *

A few microscopic slides follow:

Figure 1. A section thru a normal tonsil and parapharyngeal space.

Figure 2. A section thru the upper pole of the diseased tonsil. Epithelium and subepithelial layers of lymphoid tissues are about normal. But the lateral portions of the lymphoid tissue as well as the connective tissue and muscles of the pharynx are changed into an abscess. In the septa of the tonsil are a large number of small thrombosed veins, which do not contain bacteria. Single cocci may be seen in the lymph vessels. The internal jugular vein at this point is normal.

Figure 3. This is a section thru the abscess. Almost all of the veins which surround it are thrombosed and contain streptococci. The greatest extent of the abscess is at about the middle of the tonsil.

Figure 4. Shows a thrombosed vein with destruction of its wall at one point, as the stain indicates that the cocci have penetrated the wall.

Figure 5. A section thru the inferior third of the tonsil. The abscess is quite small and lies in the parapharyngeal space beyond the intact musculature of the pharynx. The thrombosed internal jugular vein is to be seen. A lymph gland close to the vessels shows no pathologic change.

In the lower sections, the tissue is nearly normal and only the thrombosed internal jugular vein is to be seen.

This series of sections indicates a primary thrombosis of the tonsillar veins; the first thromboses appear with the occurrence of the abscess. The numerous thrombosed veins in the upper part of the tonsil contain no bacteria, which are seen only below a certain level. The bacterial invasion must originate from the abscess, because of the fact that the remaining tonsillar tissue with its vessels is perfectly normal even in these sections. Bacteria are also to be found in the lymph vessels of the parapharyngeal space. The lymph glands are intact and in the marginal sinuses are occasionally filled with bacteria.

Only the most important points are here touched upon, and additional proof is furnished by a further series of cases combined with records of patients and operations in the clinic of Profesor Hajek in Vienna. They lead to the following opinions:

There is no doubt that venous thromboses arise in a large number of cases, probably even more frequently than is discovered by the pathologist during gross autopsy. These are mostly primary thromboses of the tonsillar veins, but primary thromboses of the internal jugular vein are also possible.

However, this disputed question loses much of its importance when, on the basis of the findings, another question is brought to the foreground: viz.,

Are tonsils and thrombosed veins to be regarded at all as the main focus of infection, the elimination of which is above all to be sought?

My examinations show, that in the great majority of cases, abscesses are found in the immediate neighborhood of the tonsil, thrombosis of the veins much more seldom, venous thromboses without abscess formation, almost never. In addition, if you find, that with the appearance of the abscess in the several sections, the surrounding veins almost without exception are thrombosed, then the supposition of a causal connection between abscess formation and thrombosis occurrence is not to be overlooked. Both thrombosis and bacterial invasion originate from the abscess.

Thus, the destructive effect does not come from the tonsil but from the abscess and from the spread of the infection into the lymph vessels of the neighborhood. The thromboses are not in themselves the fatal factor, as such thrombosed veins can probably be found in any case of abscess examined histologically. The patient's fate, then, does not depend on the condition of the vessels, but on whether or not we are able to overcome the abscess. Therefore, the question of operation on the thrombosed veins is of less importance.

Undoubtedly there are cases also in which no abscess formation arises, and where there is no spreading of the infection along the lymph channels, but where direct infection of the organism occurs by way of the blood stream. But these cases are far in the minority and may be regarded as exceptions.

* * * *

From these brief considerations we may state the follow-

ing facts of importance in helping to explain the pathogenesis of posttonsillitis pyemia:

In the series of histologic sections thru the tonsil and the neighboring region, thrombosis of the tonsillar veins occurs as a rule; primary thrombosis of the internal jugular vein is rarer. The jugular vein in its upper portions is free from thromboses; the glands are intact or contain cocci in their marginal sinuses. Retrograde thromboses may also occur.

Thrombosis formation is not synonymous with bacterial invasion, nor does the thrombus always indicate the route of infection. A large number of the capillaries and even the larger veins can be thrombosed, but at only a few places does invasion by the bacteria take place, and this not in the tonsil itself, but outside of it, from the abscess and the lymph vessels of the parapharyngeal space. There are also further cases where a thrombus reaching even into the internal jugular proves absolutely sterile.

In the great majority of cases of posttonsillar pyemia, abscess formation occurs and invariably with the same localization, viz., in the anterior part of the parapharyngeal space, often multiple, connected only by the thrombosed parts of the veins. In some places, abscesses and thromboses are not to be differentiated.

Cases without abscess formation, in which direct infection from the blood stream of the tonsils takes place, make up only a small per cent of the examined material. A few other cases also speak for the possibility of infection from a suppurating gland.

From the operative results, we conclude that where the abscesses could be completely drained, healing was obtained even without ligation or resection of a vein; where the abscess could not be completely drained, the case terminated fatally even after eventual ligation and resection of veins. These results combined with the results of histologic examination allow us to form the following conclusion:

Abscess formation and the breaking of the infection into the lymph vessels of the parapharyngeal space are to be regarded as the sepsis focus.* The thromboses play a minor role.

On the basis of this conclusion, elimination of the focus

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*Sepsisherd (Schottmüller).

must be regarded as the primary aim of the treatment; all operations on veins are of secondary importance.

Opening and drainage of the parapharyngeal space by the external route, after preventive mediastinotomy, therefore is the method of choice. Thrombosed veins, if easily recognized and accessible, may at the same time be ligated and resected.

No rule can be laid down for choosing the exact moment for operation; the tendency should be to operate rather more often and early than once too late, and operations should not be delayed until pus is found in the vessel sheath.

Posttonsillitis pyemia in children is one of the rarest of conditions.

DISCUSSION.

DR SAMUEL SALINGER, Chicago, Ill.: Since 1925, when Zange published a paper on this subject, there has been a steady increase in original observations, which have added greatly to our knowledge of the sepsis following angima. The pathology of the condition seems to be pretty well worked out, altho there are still a few minor points in which opinions differ. The topic that engrosses our attention mostly now, is the practical application of this work in histopathology. Zange attempted to divide the cases into three groups according to the severity of the symptoms, but failed to draw the lines of differentiation closely enough to be of any value as a guide to treatment. His mild cases got well and his severe cases died, and that's all there was to it.

The question is this: Shall we open up and ligate all the accessible venous channels, or shall we simply attempt to drain? In most cases, we have a peritonsillitis, with or without pus formation, and it is no mistake to open widely and attempt to remove the original focus. But in the severe cases this is not enough. The venous channels are invaded early, and lung metastases occur often with the first chill. Hans Clause showed in twelve autopsies, that eleven had pulmonary infarcts and multiples abscess. He pointed out that this complication follows much earlier than it does from otitic infection, and even when the jugular and the facial veins were ligated, death occurred in some cases from lung complications. It would seem, however, that the earlier the surgical intervention, the better the chances for recovery. One thing is sure: These cases can become rapidly virulent, and it is only by close observation that we can judge as to when to intervene and how far to go.

DR. O. JASON DIXON, Kansas City, Mo.: I am very grateful to Dr. Waldapfel for this very interesting discussion. I have in my hands a manuscript that I wrote on this subject a few weeks ago, but which was returned to me with one of these ultracourteous letters thanking me for my interesting contribution, but inferring between the lines that there probably was no such disease. I thought I had something original before I sent it in, but later I found that several other men had written about this disease.

A nurse, eighteen years of age, was admitted to my service with tonsillitis, her first attack. It was an ordinary tonsillitis, but about the third day she had a chill; then she said the left side of her neck was

sore, and she had some pain in the chest. I asked the internist to go over her, and he reported negative findings in the chest, but I said I thought she had pneumonia. She looked like a very sick girl, so we X-rayed her, but found nothing. She kept on having chills every day. The third day I made a diagnosis of thrombosis of the left internal jugular vein, and took up with a general surgeon the matter of ligation, but he did not seem to take to the idea, and the girl died on the seventh day.

Autopsy revealed thrombophlebitis of the internal jugular vein, and rather negative findings in the tonsils. No abscess, but a breaking down of the wall of the internal jugular vein. The pathologist, Dr. Ferdinand Helwig, had this comment to make:

At the autopsy there was found a swelling of the neck on the left side; nothing of interest otherwise. The abdominal organs were negative, except for some swelling of the liver and kidneys. In the chest there were fairly large abscesses which were covered with exudate. Histologic examination revealed early embolic abscess. The left internal jugular vein presented a very interesting picture. It was filled with grayish-black thrombotic material which distended it to the angle of the jaw. Streptococci were found in the pus cells; tonsils showed no acute process.

Within three weeks I had another patient who had tonsillitis, and had had four incisions for peritonsillar abscess. Nothing was found. This was at three in the afternoon. She complained of headache and had all the clinical signs of meningitis. She died the following day.

I told Dr. Helwig about the case and suggested that he be present at the autopsy. He got there as they were closing. They had not dissected the internal jugular vein. After this vein had been explored the same lesion was found as in the former patient, hence our conclusion was identical with the other case.

Dr. Richard Waldapfel, Vienna, (closing): In regard to Dr. Salinger's discussion, I have nothing to say. I am of the same opinion.

Dr. Dixon stated that the diagnosis of the general practitioner was pneumonia. That is what we find everywhere—that many of these cases which we now call posttonsillitis pyaemia have been diagnosed as pneumonia and other things. But we find in most cases that the point where the trouble started was the tonsil. In many cases, we did not see anything in the tonsil. Therefore, it is a modern disease. We did not know anything about posttonsillitis pyemia five or ten years ago.

He also said this case shows infection in the lymph glands. In the Presession Volume, we did not mean that the thing we want to find is an abscess. In many cases, all they need is deep drainage. The second case had some infection of the cerebrum, he thinks. A very interesting publication by a doctor of the Hajek Clinic shows that what we showed here downwards, is also possible upwards, and I think this other case belongs in this group. This infection goes upwards along the trigeminus, and many of these cases have as a symptom high temperature. If a patient after paratonsillitis has high temperature, do not forget to think of infection going upward to the cerebrum.

I expected a little resistance from the pathologic side. I have not so much experience in histologic work on this question, but even if it had been done, it would show that the condition in the mastoid and here are absolutely different.

BRONCHOSCOPIC STUDY OF ASPIRATION IN TONSILLECTOMY.

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HOUSTON, TEXAS.

Since Richardson, in 1912, called attention to the occurrence of lung abscess following tonsillectomy, Scudder, Whittemore, Moore, Singer and Graham, Cutler and Schlueter, Hedblom, Fisher and Cohen, Chipman, Homans, Porter, Lord, Manges, Lockwood, Wessler, Jackson, Tewksbury, Clayton, Simpson and Noah, Clerf and a number of other investigators established the fact that a considerable percentage of nontuberculous abscesses of the lungs follow operative procedures on the upper air passages, particularly tonsillectomies. Schlueter and Weidlein tabulated 1908 cases of lung abscess, and found that 29.61% followed operations, half of which were tonsillectomies.

There is still a definite divergence of views concerning the mode of occurrence of posttonsillectomy lung abscess. Jackson, Fetterolf and Fox, Cutler and his coworkers, Homan, Schlueter and Weidlein and others maintain that the abscesses are caused by infected emboli from the peritonsillar tissues; Myerson, Lynah, Coakley, Voorhees, Smith, Alton and Wellwood, Aschner, Lockwood, Lemon and Borden believe that the abscess is due to direct aspiration of infective material during the operation. It is obvious that until the etiology is definitely known, our prophylactic measures will be lacking in faith and of necessity half hearted.

Cutler and his coworkers, Schlueter and Weidlein, base their adherence to the embolic theory on their failure to produce experimental aspiratory abscess in dogs, while they have succeeded in developing acute, rapidly healing abscesses by injecting into the jugular vein a mass of bacteria enclosed in a capsule, to prevent dissemination over a large pulmonary area. Homans argues against the aspiration theory, because the majority of lung abscesses are located in the upper lobes of the lungs. Many

observers feel that only the embolic theory can explain the occurrence of lung abscess following tonsillectomy under local anesthesia. In favor of the aspiration theory, Lockwood states that embolic abscesses are usually pyemic in their course, while posttonsillectomy abscess is chronic. Aschner, from a pathologic study of ten posttonsillectomy cases, has shown that the abscess cavity is a direct continuation of a bronchus, and that the cavity is lined with epithelium which is continuous with the bronchus. Lambert and Miller made a bacteriologic study of ten cases of lung abscess at the time of the operation; they report the uniform presence of anaerobic bacteria, the group which is often found in the mouths and throats in pyorrhea, and particularly in the tonsillar crypts. Smith produced experimental pulmonary lung abscess in 20% of mice, guinea pigs and rabbits by introducing intratracheally material obtained from the teeth of persons with pyorrhea. Myerson found blood bronchoscopically in 70 out of 100 cases of tonsillectomy. Allen produced lung abscess in three out of seventeen dogs by intrabronchial injection of warm pus from patients with chronic lung abscesses. Crowe and Scarff produced lung abscesses in dogs by bronchoscopic introduction of pledgets of cotton soaked with fresh scrapings of human pyorrhea cavities. To these, we wish to add our bronchoscopic and esophagoscopic observations of one hundred cases of tonsillectomy under general anesthesia, and six cases under local. A preliminary report of these investigations was made before the Texas Medical Society in 1927.

The youngest patient examined was three years old, and the oldest thirty-five; the majority were younger than twelve. All had the routine preliminary examinations and were the usual surgical risks. The operative time varied from a few minutes to three-fourths of an hour. The tonsils were enucleated by dissection and snare or the Beck-Sluder guillotine. The anesthesia was administered by the hospital anesthetist, and consisted of nitrous oxid and oxygen induction, followed with ether by the open method, till the patient was sufficiently relaxed to permit the introduction of the mouth gag; a warm ether spray was then maintained as necessary thruout the operation. Suction was maintained continually by an assistant. When the operation was completed and the pharynx dry, the patient was reanesthetized if necessary, and the examination made with the Kahler Leiter bronchoscope.

In the last seventy cases, the tube, after being withdrawn

from the trachea, was introduced into the esophagus; if the stomach contents did not run out thru the tube, the inner tube was passed thru the cardiac orifice of the stomach.

The first sixty-seven cases were operated on under the anesthesia ordinarily seen in tonsillectomies; the patient was well relaxed, the pharyngeal reflexes were abolished, there was no gagging, straining or resistance to the tongue depressor. The first thirty cases were bronchoscoped; blood was found in the bronchi in all cases; the next thirty-seven cases were bronchoscoped and esophagoscoped; there was blood in the tracheobronchial tree and none in the stomach in thirty-three cases; blood in lungs and stomach in two cases; blood in neither in two cases—these last two were bloodless operations in very young children. The next case was a twelve year old girl, on whom Dr. S. P. Pulliam did a Beck-Sluder tonsillectomy; the anesthetist seemed unable to anesthetize her deeply enough for the ordinary smooth operation; she had a profuse hemorrhage, requiring several sutures, and because she was gagging and vomiting, the operation lasted longer than usual, and her mouth was full of blood in spite of suction. Instead of finding her bronchi full of blood, as we expected because of the amount of blood in her throat, the bronchoscope showed the lungs free of blood; the stomach contained a large quantity of blood. Having previously observed the effect of position, ether spray, and amount of hemorrhage on the blood content of the lungs, we felt certain that this freedom of the lungs from blood could be due only to the incompleteness of anesthesia. The succeeding cases operated upon under light anesthesia presented the same endoscopic findings.

These phenomena led us to believe that it is possible to eliminate aspiration of blood and secretions into the lungs during tonsillectomies, by operating with the swallowing reflex unabolished. Mosher in an X-ray study of the movements of the tongue, epiglottis and hyoid bone in swallowing, demonstrated that in the act of swallowing, the epiglottis rises with the base of the tongue, becomes applied to it, and finally is forced back against the posterior pharyngeal wall; the approximation of the tips of the arytenoids to the base of the cushion of the epiglottis, and the sphincter like action of the aretynoepiglottidean folds serve to make the closure of the larynx complete. Normally, the larynx is an open space with air going in and out; the esophagus is closed at the cricopharyngeal con-

striction, which, as pointed out by Jackson, is similar to an incomplete sphincter at this point; when the sensory nerve endings in the pharynx are stimulated by the contact of food or drink, the complicated act of swallowing is set in motion; simultaneously with the reflex contraction of the mylohyoid muscle and a peristalic esophageal wave, coordinated movements shut off the nasal and laryngeal openings; the soft palate shuts off the nasal opening, and the adductors of the larynx and epiglottis close the larynx, causing a temporary inhibition of respiration.

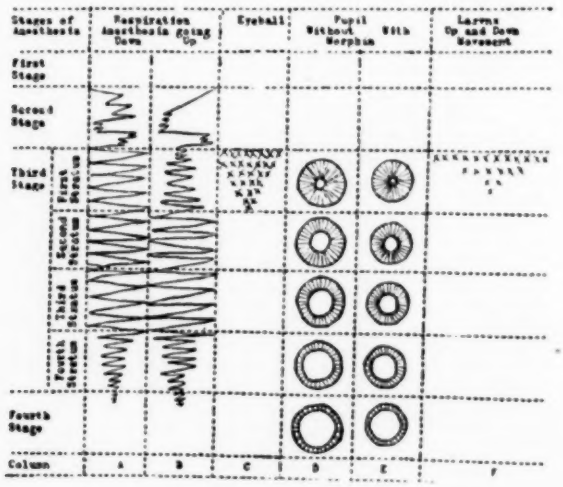


Fig. 1. Anesthesia Chart (Guathmey).

The closure of the larynx, therefore, is initiated by the swallowing reflex. To maintain a state of anesthesia with the pharyngeal reflexes intact, is not ordinarily easy; as may be seen from the schematic chart of the various stages of anesthesia, the third stage is the stage of surgical anesthesia; in the second stage there is vomiting and retching; in the upper border of the third stage, when the eyeballs are still oscillating, and the larynx moving up and down, the swallowing reflex is present; there is some resistance to the tongue depressor, and the operation is somewhat more difficult than when the patient is absolutely relaxed. If the anesthesia has not quite reached this stage, there is vomiting, and if it passed somewhat beyond it, the pharyngeal reflexes are abolished.

Tabulating our results, we find that of 79 cases operated on under deep anesthesia, 74, or 94.9%, had blood in the lungs bronchoscopically. Of 21 cases operated on under light anes-

thesia, four, or 19%, had blood in the tracheobronchial tree, a much smaller percentage as compared with the percentage under deep anesthesia. Of 49 cases under deep anesthesia, 4, or 8.16%, had blood esophagoscopically in the stomach contents. Of 21 cases operated under light anesthesia, 19, or 90%, had blood in the stomach contents.

The factors investigated with regard to their influence on the amount of blood aspirated are:

1. The amount of hemorrhage—obviously the more blood there is in the mouth, the more there is to be aspirated. In the cases where the operations were bloodless, there was no blood in the lungs or in the stomach, even under moderately deep anesthesia.

2. Position of the head—even with the head of the patient lowered 45 degrees, there was always some blood found in the lungs in the cases which were operated on under deep anesthesia; however, the amount was decidedly less than when the patient is operated on in the horizontal position.

3. The elimination of the ether spray had no effect whatever.

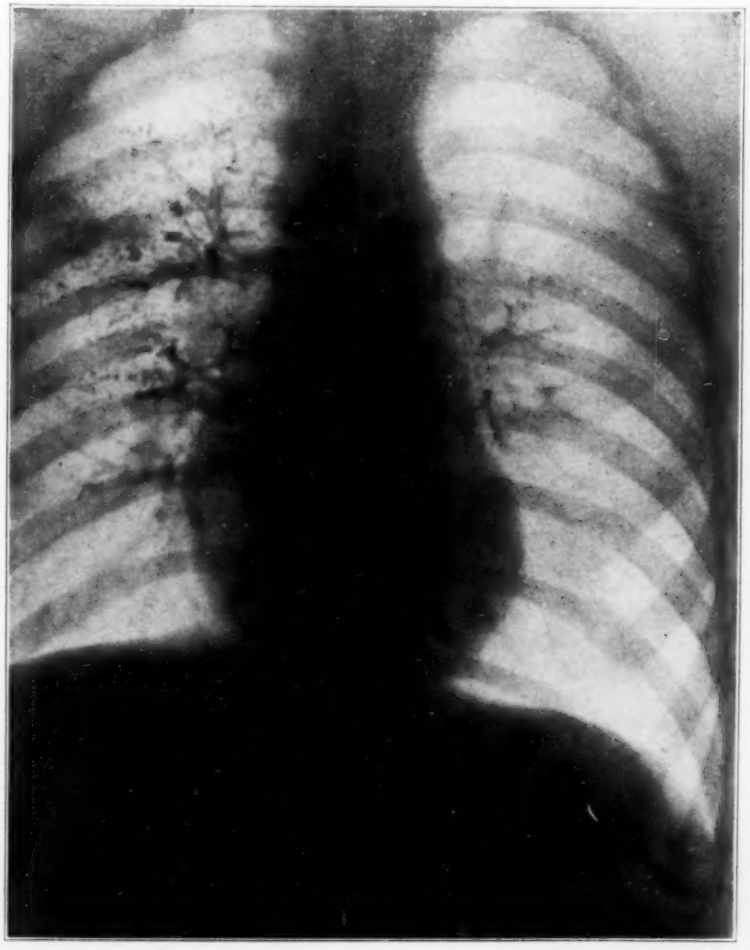
The outstanding phenomena of our investigations were the presence of blood in the lungs and its absence in the stomach, or vice versa, and the fact that it depended almost entirely on the depth of anesthesia. These results are contrary to those Lemon obtained in his experiments on dogs; he found that aspiration in dogs occurred regardless of the depth of anesthesia, and depended chiefly on position; that aspiration diminished with the downward inclination of the head and disappeared entirely in the Trendelburg position.

Our findings also prove fallacious the oft repeated statement seen in the literature, urging as a prophylactic measure against aspiration in tonsillectomies an anesthesia abolishing the pharyngeal reflexes. Our investigation convinced us that the very lightest anesthesia which will permit of an operation is the anesthesia of choice, to avoid aspiration of blood and infective material into the lungs. If posttonsillectomy lung abscess is of aspiratory origin, the offending agent is probably the bacteria laden material squeezed out from the tonsils with the grasping forceps or tightening snare; it appears to us, therefore, that the tonsils should be removed under a light anesthesia, even if later it may be necessary to deepen the anesthesia for suturing.

Having found the astonishing frequency of aspiration of blood during tonsillectomies, we wanted to see if possible by what mechanism the lungs free themselves of this accumulation. Bullowa and Gottlieb found, in their experiments on dogs, that the tracheobronchial tree empties itself in twelve to fifteen minutes by a bellows or accordion like action, and a peristaltic wave of the bronchial musculature. A. L. Brown and E. Archibald report that a mixture of lipiodol and fluid sputum remained in the lungs of animals for as long as twenty days, particularly in those animals in which cough caused a deeper penetration. Fried and Whitaker saw iodized oil in the lungs of cats after four months. Archibald and Brown believe that iodized oil is absorbed from the bronchial and alveolar lumina, and that in the majority of cases the shadow persists only a few weeks, but in some cases as long as fifteen months. In ten cases, after the tonsillectomy was completed, we injected 4 c.c. of lipiodol intratracheally thru the bronchoscope, and observed the patients fluoroscopically. At first, the lipiodol is seen scattered diffusely thru the lungs, mostly at the periphery and in the upper and middle lobes of the right side; a snake like, animated, twisting movement of the lipiodol shadows is seen, sweeping the lipiodol toward the larger bronchi, where it collects in lumps; there is a definite upward movement, and lipiodol is seen coming up the trachea, and several times we have seen a lipiodol shadow come up, jump over the epiglottis, descend the esophagus and enter the stomach; as the patient wakes, the lipiodol may be seen returning up thru the esophagus and is vomited. Apparently, most of the aspirated material is rapidly eliminated from the lungs and passed into the stomach. Allen, in his experiments on dogs, thinks that "trapping in" of the infective material in some of the smaller bronchi plays a dominant role in the development of lung abscess. The following slides were made from the X-ray plates of two patients injected with lipiodol:

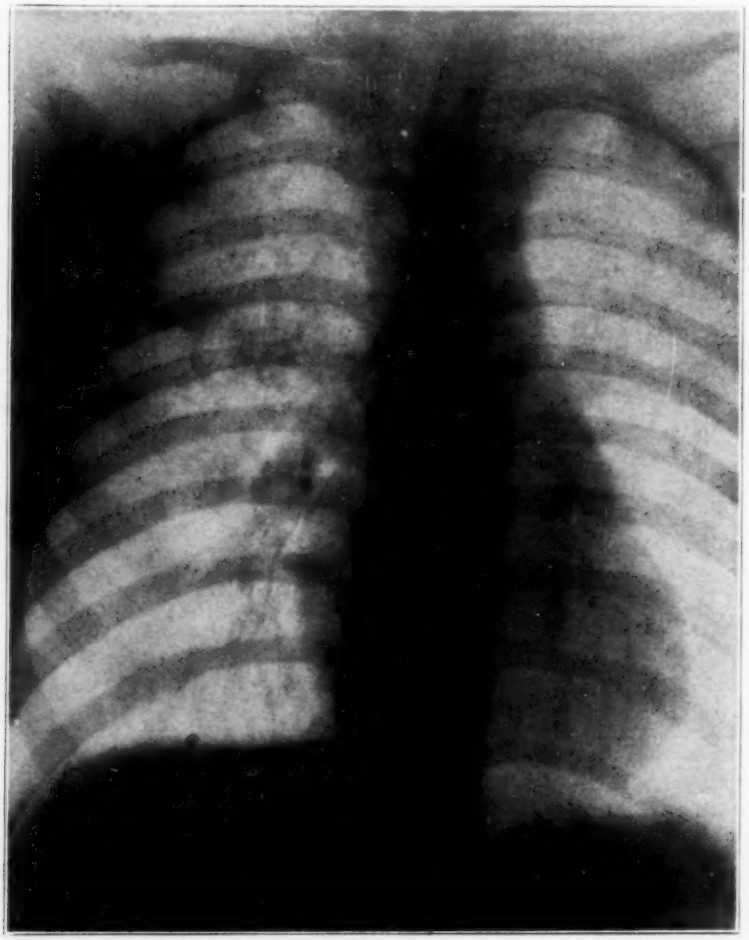
Case 1. P. J., 5 years old; seven minutes after the operation, 4 c.c. of lipiodol was injected intratracheally. Plate 1, taken eleven minutes after the injection: A large quantity of lipiodol is seen in the upper and middle lobes; it is distributed diffusely thru the finer bronchi; the large bronchi in the middle and upper lobes are well outlined, and the trachea is outlined in its lower third. A small quantity seen in the left lung about the hilus; none in the esophagus or stomach. Plate 2, taken fourteen minutes later, or twenty-five minutes after the injection:

The lipiodol is seen collected in large clumps in the larger bronchi, mainly in the upper and middle lobes; entire trachea and esophagus outlined, and scattered masses seen at various points in esophagus, and some of it seen in the stomach. Plate 3, thirty-two minutes after the injection: Patient had a coughing spell between the taking of plates 2 and 3. Quantity of lipiodol in the lungs very small; much more seen in the stomach;



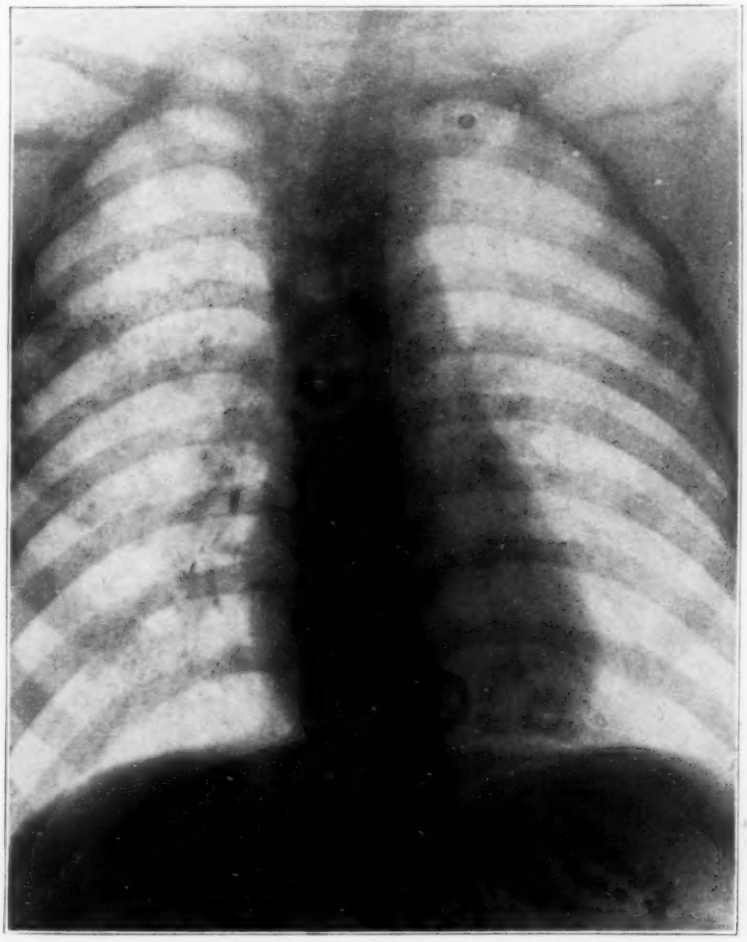
Case 1. Fig. 1. Large quantity of lipiodol in upper and middle lobes, distributed diffusely thru finer bronchi; large bronchi in middle and upper lobes well outlined, and trachea outlined in lower third. Small quantity in left lung about hilus. None in esophagus and stomach.

bronchi and trachea less distinctly outlined; more diffuse infiltration in right lower lobe, altho there is still more lipiodol in upper and middle lobes. Brown and Archibald demonstrated that cough causes a deeper penetration. Plate 4, one hour after the injection: Patient vomited in the meantime; he was waking and restless, and the picture is blurred; less lipiodol seen in periphery of lung and more in the bronchi and trachea; less lipiodol seen in the stomach.



Case 1. Fig. 2. Lipiodol collected in large clumps in the larger bronchi, especially in upper and middle lobes. Trachea outlined, and scattered masses seen at various points in esophagus. Some lipiodol in the stomach.

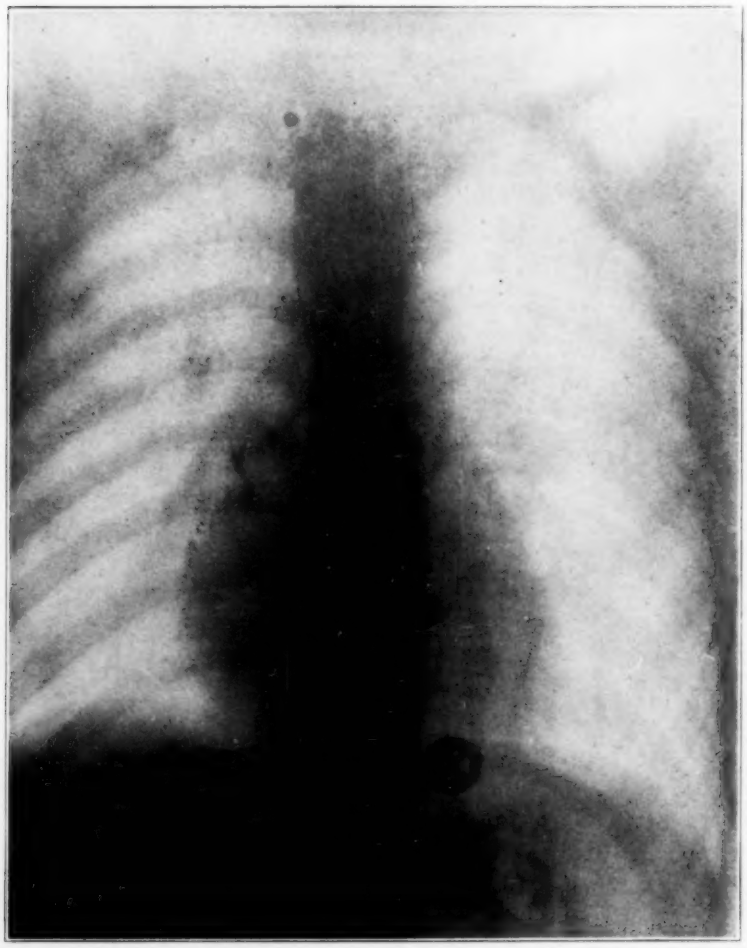
CASE 2. C. M., five years old; 4 c.c. of lipiodol injected intratracheally in the horizontal position, immediately after the operation. The child was lightly anesthetized, and when she reached the X-ray room, she was vomiting. Plate 1, twelve minutes after the injection: Diffuse infiltration of right upper and middle lobes; small quantity in right lower lobe and in left lung about the hilus. The larger bronchi and trachea well outlined; lipiodol seen in esophagus and stomach. Plate 2, six minutes later: Beginning concentration of lipiodol in larger



Case 1. Fig. 3. Bronchi and trachea less distinctly outlined. Quantity in the lung very small. More diffuse infiltration in right lower lobe. Much more in the stomach.

bronchi and clumps in trachea; more of it seen in the stomach. Plate 3, ten minutes later: Concentration more marked in bronchi nearest trachea and large masses in trachea; greater quantity in stomach. Plate 4, twenty minutes later: The child had a vomiting spell; fluoroscope showed the lipiodol coming up the esophagus; less lipiodol in the stomach, very small quantity diffused in the lungs, and some large clumps in trachea.

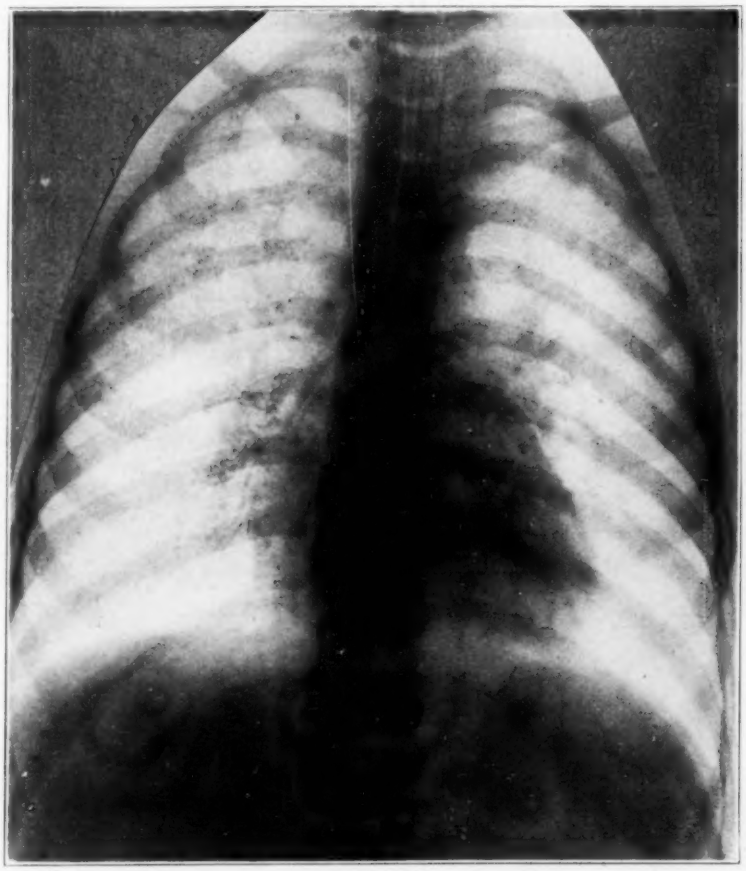
These observations agree with those of Reinberg, who introduced opaque substances into bronchial fistulae, and observed



Case 1. Fig. 4. Less lipiodol in periphery of lung, and more in trachea and bronchi. Less in the stomach.

fluoroscopically the lungs, as they empty themselves by what he, too, describes as an active peristaltic movement.

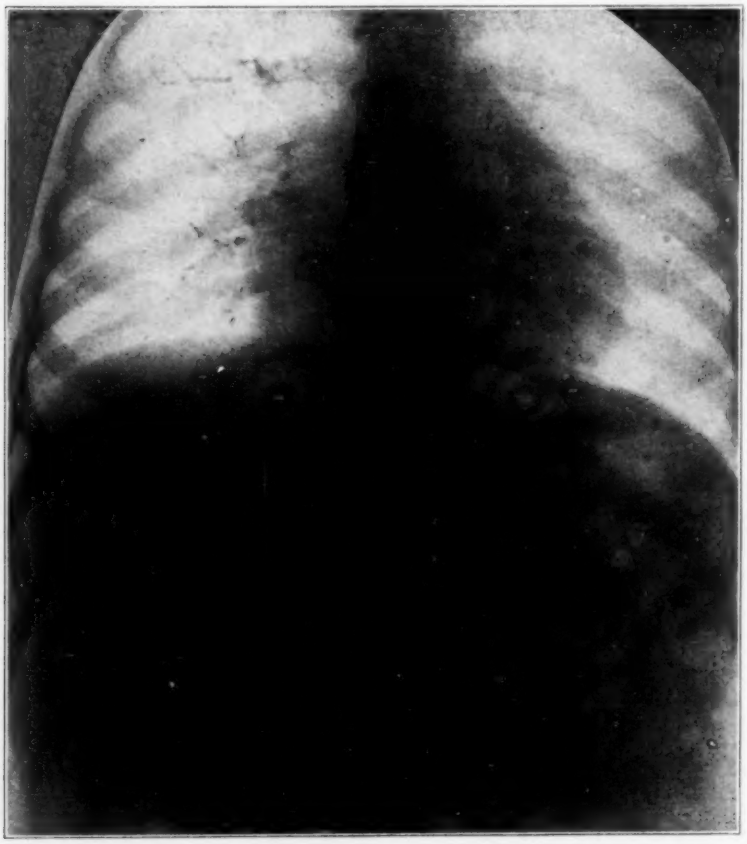
Another phenonenon that recurred with sufficient frequency to force itself upon the attention, was the fact that lipiodol injected intratracheally in the horizontal position tends to flow into the upper and middle lobes of the right side, while, if injected in the sitting or semirecumbent posture, it tends to flow more into the lower lobes. This may explain why the majority of posttonsillectomy abscesses are located in the upper and middle lobes of the right side, while the majority of aspirated



Case 2. Fig. 1. Diffuse infiltration of right upper and middle lobes. Small quantity in right lower lobe and in left lung about hilus. Larger bronchi and trachea well outlined. Lipiodol present in esophagus and stomach.

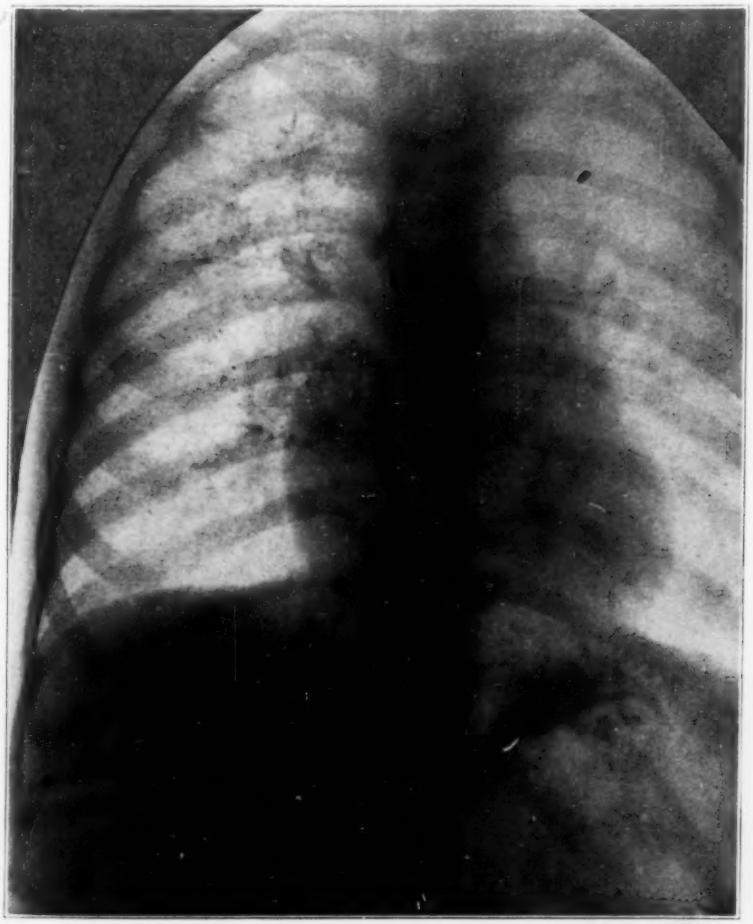
foreign bodies are located in the lower lobes. Foreign bodies are usually aspirated in the upright position, while material aspirated during tonsillectomy is aspirated in the horizontal position. Lockwood states that aspiration abscesses occur about three times as often on the right side as on the left, and about two and one-half times as often in the upper and middle lobes as in the lower. Clerf, in a study of fifty-eight cases, reports 63% in the right lung and 37% in the left; in the majority of cases the upper and middle lobes were involved.

In our six local cases, anesthetized by the injection of novocain thru the anterior pillars into the paratonsillar tissues, two cases had blood in the tracheobronchial tree. Aspiration



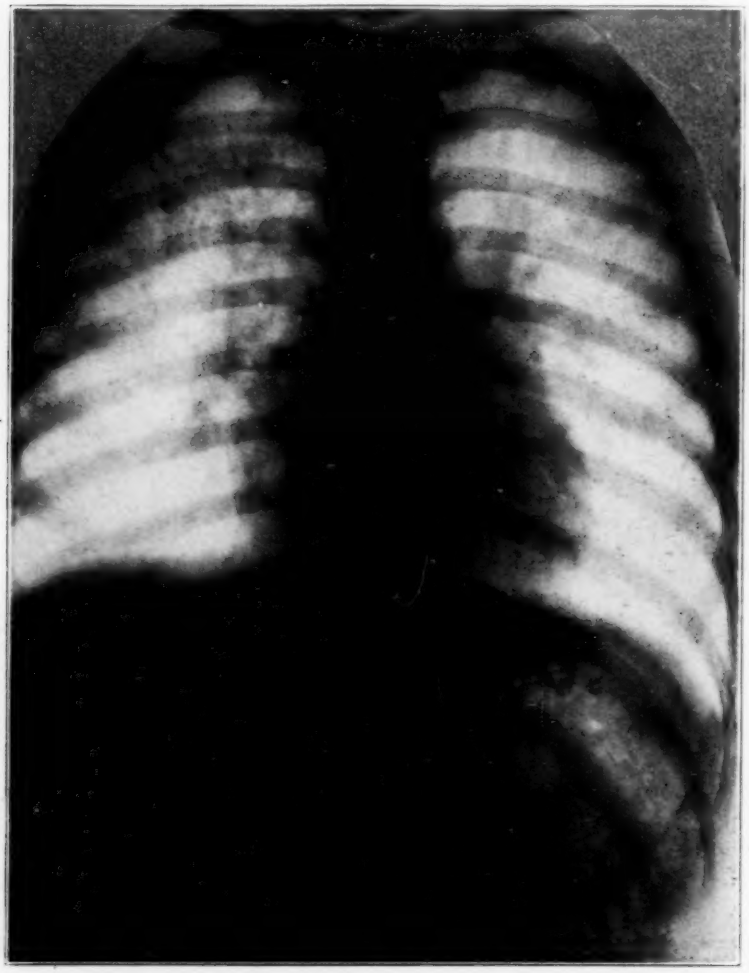
Case 2. Fig. 2. Beginning concentration of lipiodol in larger bronchi and clumps in trachea; more in the stomach.

occurs during local tonsillectomies, altho these observations do not altogether confirm the observations of Alton and Wellwood, who gave five local tonsillectomy cases lipiodol to swallow, and saw fluoroscopically the lipiodol pass into the trachea and bronchi instead of into the esophagus, in every case. Pommerence, in a study of the sensory areas which will initiate the swallowing reflex, found that "the anterior pillars of the fauces are the most effective in instituting the swallowing re-



Case 2. Fig. 3. Concentration more marked in bronchi nearest trachea; large masses in trachea; greater quantity in stomach.

flex." It is probable that the injection of the anterior pillars in local anesthesia interferes with the proper initiation of the swallowing reflex. The number of cases is too small to justify definite conclusions. It would be interesting to obtain accurate statistics of the lung abscesses developing following local tonsillectomies, with regard to their method of anesthesia, operative position and location in the lungs.



Case 2. Fig. 4. Less lipiodol in the stomach. Very small quantity diffused in the lungs, and some clumps in trachea.

Conclusions.

1. Aspiration of blood occurs in 90% of tonsillectomies performed under deep anesthesia.

2. Aspiration can be avoided by operating with the swallowing reflexes intact.

3. The lung eliminates the aspirated blood by a peristaltic wave, assisted probably by the action of the ciliated epithelium and cough.

4. The location of posttonsillectomy abscesses points to aspiration as their cause.

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DISCUSSION.

Dr. L. A. Schall, Boston, Mass. The paper of Dr. Daily deals with both fact and theory. He proves the fact that under deep anesthesia blood can be found in the tracheobronchial tree much more frequently than under light anesthesia.

The theory deals with the causation of postoperative pulmonic abscess, and this theory always makes an interesting controversy. Perhaps there is some truth in all the theories. Personally, I am inclined to be in sympathy with the aspiration theory, but it does not explain to me conclusively the postpulmonic abscess seen after operations other than those in the upper respiratory tract. In justice to our patients, we should adopt any and every measure which adds to the safety of the operation,

and if you can operate as skillfully and as thoroly in the presence of an active gag reflex, then the fact has been shown to outweight the theory, and our patients should be given this added measure of safety.

Dr. Gabriel Tucker, Philadelphia, Pa.: I wish to compliment the Drs. Daily on their excellent presentation. Their investigations and conclusions give us a rational basis for effective prophylaxis in postoperative pulmonary complications, by preventing inspiration of infective material. Their lipiodol studies were most interesting, and helped to explain to us the greater frequency of occurrence of postoperative and posttonsillectomic abscess in the upper lobes of the lungs.

Our bronchoscopic observations of the lungs in postoperative pulmonary abscess cases lead us to agree with the authors in the belief that probably not all cases are of inspiratory origin. The local appearances on bronchoscopic examination in pulmonary abscess cases of the same duration, present pictures so different, that it would seem that both lesions were not produced in the same way. In one group of cases, there is definite evidence of infection in the bronchus, foul, blood stained pus, local edema and swelling in the mucous membrane, definitely localized in a small area of bronchial distribution. An infection of inspiratory origin would account for this local lesion. In the other class of cases, there is very little local reaction, but pus is found coming profusely from branch bronchi. There is no local evidence of inspiratory infection. Hematogenous infection would fit better into the local findings in these cases. However, the possibility of inspiration of infective material into the alveoli of the lung without local reaction in the larger bronchi must be considered.

Until such time as the theory of hematogenous origin of pulmonary abscess has been disproven, our prophylactic measures should take into consideration the blood stream route of infection as well as the inspiratory route.

While we are not certain as to the exact pathway of infection in some cases, our experience in the treatment of postoperative pulmonary abscess has given us a large number of cases of cure by bronchoscopic aspiration. We conclude, therefore, that in postoperative pulmonary complications, the earlier bronchoscopic treatment is instituted, the better the prognosis for cure, provided, of course, that the primary surgical condition or pleural involvement does not contraindicate the procedure in the individual case.

Dr. H. H. Vail, Cincinnati, O.: There have been 400 cases studied bronchoscopically following a tonsil and adenoid operation under general anesthesia, (ether). Dr. Myerson has bronchoscoped 200 cases, the Drs. Daily bronchoscoped another series of 100, and recently, in the Cincinnati General Hospital, another group of 100 cases has been bronchoscoped and observations made as to the presence of blood in the tracheobronchial tree. Briefly, Myerson found in his first series, that 79 patients had aspirated blood; in the second series, that 76 patients had aspirated blood. Daily found that 78 patients had aspirated blood, and we found that 62 had aspirated blood. This just means that more than a majority of our patients under ether following tonsil and adenoid operation must aspirate blood. Our series was done under the ordinary form of anes-

thesia, the patient being put in a comfortable state of anesthesia, so the work could be done in a safe, rapid fashion, and yet 62 per cent of these patients had blood in the tracheobronchial tree. Some of the patients, who were under so lightly as to be gagging and vomiting, showed blood in the tracheobronchial tree.

We felt it was a question of the amount of bleeding at operation. Of course, it is obvious that if you have a bloodless operation, you will not get blood in the tracheobronchial tree. We know that in every clinic there are thousands of tonsillectomies done in the course of a year, and very little in the way of postoperative pulmonary complications can be laid to the operation. I think we have overlooked the fact, that in these children who come for tonsil and adenoid operation, there may be nasal sinusitis with a pulmonary infection before we operate; and I would suggest, that before we lay too much stress upon the aspiration of blood, we examine these patients a little more carefully as far as their nasal sinuses and chest conditions are concerned, before we subject them to operation under general anesthesia.

Dr. Samuel Iglauer, Cincinnati, O.: Lung abscess after tonsillectomy, as Dr. Vail has just said, does not occur very frequently, still you remember the statistics of Moore, who found it occurred once in every twenty-five hundred to three thousand tonsillectomies. It is, therefore, a real danger to men who are doing tonsillectomies.

I cannot go into the subject of the aspiration theory very fully, but the vast majority of lung abscesses that occur in surgery, not only tonsil surgery, but general, occur after operations about the mouth. This has been shown conclusively by the work of Lord, of Boston.

In regard to pyemia, I would like to refer to Dr. Waldapfel's paper. If we had a lung abscess as a result of infection thru the venous channels, we would not have solitary lung abscesses but the thing he has described, multiple infarcts in the lung, whereas our pulmonic abscesses are usually single. Furthermore, if these abscesses were not often of inspiratory origin, Dr. Tucker would not be able to relieve them by the bronchoscope.

Against the inspiratory theory, there is an argument that lung abscess does not occur as often after tonsillectomy under local, as under general anesthesis. I examined fifty cases after removing their tonsils under local, and found that in 40 per cent there was some blood in the trachea, of course not nearly the amount as under ether. The fact that a patient has had a local anesthetic, does not preclude the aspiration of blood. Therefore, the aspiration theory holds for local anesthetic as well as general. (Iglauer, Annals of Otol. Rhin., and Laryn., March, 1928.)

Ochsner tried to settle this question by anesthetizing the throat as for tonsillectomy, and then telling the patient to swallow lipiodol. He found that in all five cases the lipiodol went into the lung. I tried this same experiment in five cases, but told the patient to expectorate instead of swallowing, and I found some lipiodol in the lung in but two cases, but most of it in the stomach in all cases. After anesthetizing the tonsil region, one probably abolishes the reflex which controls swallowing, and some of the lipiodol and blood may enter the lung.

One of the chief factors in the production of lung abscess is retention of secretion. This has been shown experimentally by Crowe. He could

not produce lung abscess in the dog until he put a piece of cotton down and imprisoned the bacteria. He then produced experimental lung abscess in dogs.

Dr. Richard Waldapfel, Vienna: After the clinical experience of the Viennese clinic, one must be grateful to Dr. Daily for throwing light on this question. All the American doctors who have been to our clinic in Vienna have seen how we do tonsillectomies. It is certainly different from the way it is done here. It is no rare occurrence to hear an American doctor say, "In America, we would not be allowed to operate as you do here." And how do we operate? In children, we operate with the patient sitting up, and give it just the necessary anesthesia, and no more. In fact, in most cases the patient is awake by the time the operation is finished.

We do not know the complication of lung abscess after tonsillectomy! This is, therefore, the substance of our reply to the American doctors, "Your method is perhaps the more esthetic one, but we prefer to be less esthetic and more safe." I do not wish to discuss the theory whether it is the blood stream or not; the fact is, that no matter where the point of entry may be, we do not have this complication. We know about it only from the literature.

We attribute our results, so far as lung abscess is concerned, to two facts: First, to the light anesthesia, for we never go beyond the swallowing reflex; secondly, to the fact that we operate with the child sitting up, which facilitates expectoration.

I am glad to find proof of our clinical experience in this very scientific presentation of Dr. Dailey.

Dr. Ray K. Daily, Houston, Tex., (closing): Dr. Schall questions the place of aspiration among the causative factors of lung abscess, because of the occurrence of lung abscess following operations other than those about the nose and throat. It is not illogical to assume that aspirations of secretions in the mouth containing the anaerobic group of bacteria, usually found in lung abscesses, is possible, and not infrequently occurs under a sufficiently profound anesthesia, regardless of the operative site. The fact that the majority of lung abscesses do occur following operations about the oral cavity, when secretions are profuse and conditions for aspiration most favorable, bears out this contention. As Dr. Iglauer pointed out, it is not far fetched to assume that infective organisms can take the route to the lungs that we take with the bronchoscope.

Dr. Vail's statement that he found just as much blood in the lungs after tonsillectomies under light anesthesia as under deep anesthesia, is at complete variance with our findings. Is it possible that what he considered as light anesthesia, was an anesthesia deep enough to abolish the swallowing reflex? In this connection, we designated light anesthesia as one that maintains the swallowing reflex intact. In our operations, with the swallowing reflexes intact, we found the lungs free of blood in the large majority of cases, and we believe, that in spite of the unsightliness of such an operation, it is of greater safety to the patient.

Aspiration occurs very frequently in tonsillectomies under local anesthesia. In one of his recent papers, Dr. Iglauer reported examinations made with the laryngeal mirror. We have since done a number of these examinations after local tonsillectomies, and then verified the findings

with the bronchoscope. It seems a very accurate method for determining the occurrence of aspiration; almost uniformly, whenever there was blood found bronchoscopically, it was seen in the larynx with the mirror. Of course, this examination gives no idea as to the quantity of blood aspirated. In several of the local tonsillectomies, after bronchoscoping them, we poured 10 c.c. of lipiodol back of the tongue and watched them with the fluoroscope. In those cases which *had not* aspirated blood during the operation, the lipiodol could be seen being swallowed into the stomach; in those cases which had, the lipiodol could be seen also passing partly into the lungs and partly into the stomach. Apparently, our method of local anesthesia—10 c.c. of 1% novocain injected into each peritonsillar fossa—sometimes interfered with the swallowing reflex sufficiently to permit of aspiration and sometimes did not. We found, that by cocainizing the pharynx thoroly with cocain, all the lipiodol introduced into the pharynx in the upright position passed into the lungs; in an imperfect anesthesia, as evidenced by the patient's ability to swallow water without coughing, most of the lipiodol was swallowed into the stomach, a small quantity at times being aspirated into the lungs.

There are still a great many questions to be answered from the standpoint of aspiration of blood and secretions during tonsillectomies. We studied the factors controlling aspiration. What of the factors controlling elimination of the aspirated material and of the power of the lung to protect itself against infection? What is the effect of anesthesia on the protective reflex of the lung? What is the effect of drugs, such as morphin and hyoscin, so frequently used in local tonsillectomies, on the eliminative mechanism of the lungs? These, and many other questions, will have to be investigated before the final word on the method of tonsillectomy can be said. We may have to revise our entire conception of the operation after a thoro knowledge of the physiologic processes taking place during its performance.

NEW PRINCIPLES IN THE CONTROL OF THE ACTIVITY OF CILIATED EPITHELIUM.*†

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It is well known that ciliated epithelium is widely distributed in many animal forms, from protozoa to higher vertebrates. In man, this tissue lines the respiratory passages—the nasal cavities and paranasal sinuses, the pharynx, the trachea, the bronchi and the bronchioles. It lines important portions of the genital system —the uterus and fallopian tubes in the female, and the proximal segments of the composite seminiferous duct system in the male. It also lines certain portions of the lumen of the cerebrospinal axis, and, according to some authorities, it constitutes the socalled brush border cells in the proximal convoluted tubules of the kidneys, but its existence in the latter location is held in dispute.

In spite of the great amount of study devoted to the subject of ciliated epithelium, our knowledge of its exact biophysical and biochemical reactions as part of the defenses of the body against noxious environmental agencies is far from being commensurate with its great biologic importance. Its importance in the defense against noxious outside agencies is suggested by the fact that in the higher vertebrates, including man, this tissue comes into far more direct relation with the environment than does any other visceral tissue. The dynamic elements of all other visceral systems are protected from environmental contact. It is true, that the perspiratory mechanism opens upon the skin surface by multiple microscopic ostia; the gland cells, however, are placed away from the surface. The endothelial system in the most superficial capillaries of the skin is also well blanketed off from the body surface. The major portions of visceral tissue, which make up the specialized cellular components of the internal or-

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†By invitation.

gans, are, of course, deeply overlaid and protected by the various somatic tissues which compose the thick body wall.

The most extensive portions, however, of the ciliated epithelial system in the body, namely, those lining the respiratory passages, are in intimate contact with the environment, being impinged upon directly by the inspired air, which is well known to be polluted usually not only by vast numbers of microorganisms, many of them pathogenic, but also by various types of dust particles in great numbers, and by other kinds of noxious materials.

Were it not for the body's defenses, the potentiality for bacterial invasions via the respiratory route, alone, would doubtless lead to more or less constant extensive and serious infection, on account of the enormous numbers of noxious bacteria more or less constantly present in the air of our homes, schools, laboratories, shops and streets. Yet, generally, our ciliated surfaces do not become infected whenever pathogenic microorganisms gain access to them. This is a matter of common observation, and it has been demonstrated experimentally by Bloomfield[1] and others, that when many types of pathogenic bacteria are brought into contact in large numbers with the upper respiratory mucous membrane of healthy persons, these microorganisms are easily disposed of within a very short time without colonization and invasion.

Bloomfield emphasizes the fact, that in the study of the etiology of respiratory infections, we have in the past overemphasized secondary factors, such as the identification of the offending organisms and the determination of its mode of transportation to the human host, but have neglected the most essential factor, namely, the respiratory surface itself, since it is only when some abnormal change or injury occurs in the host, he says, that colonization and invasion occur.

What constitutes the abnormal change in the respiratory defenses which permits infection to occur fairly frequently? Why do about a hundred million people in our country develop one to three "colds" a year? These respiratory infections lead directly to much distress and to enormous economic losses, and indirectly to resulting complications of various kinds, to serious impairment of health, and not infrequently to loss of life. I need not here mention the other well known types of serious and not infrequently fatal infections which gain entrance to the body via the respiratory portal. These diverse types of

infective organisms gain entrance to the body by breaking down the resistance in a region where the fight against invasion is carried on in a territory lined by ciliated epithelium.

The mechanisms mediating the respiratory protective functions are biophysical and biochemical. Among the biophysical, are coughing and ciliary activity. Bloomfield has mentioned another biophysical mechanism, which consists of suction currents, which free the mouth, fauces, tonsils and pharynx of bacteria that may find lodgment in these places. Since coughing does not occur until there is already considerable irritation of the mucosa, the mechanical function of cleaning the inspired air of its burden of noxious material is laid practically entirely upon the respiratory ciliated surfaces.

We are not here concerned with such biochemical processes as are related to general and local immunologic, or to phagocytic, types of defense, and which, tho highly important are, like the ciliary apparatus, insufficiently understood. Nor are we concerned with the biophysical processes described, as noted above, by Bloomfield in relation to the stratified epithelial lining of the oral and pharyngeal surfaces. Our interest in this study has been directed to certain controlling factors which regulate the rate of activity of ciliated epithelium.

The ciliary mechanism is important in another anatomic territory. The serious possibility of peritoneal infection in the female via the genital tract, which usually harbors numerous microorganisms, is normally entirely prevented, because the bacteria are unable to travel beyond the defenses which are maintained in the intervening cilia lined mucous surfaces.

It is generally recognized that our knowledge of the manner in which these vitally important defenses are maintained is wholly inadequate. More light as to precisely how these defenses are mediated in cilia lined territory is much to be desired. We must admit, that in spite of the imposing array of facts and opinions that have been woven about the modus operandi of the respiratory protective mechanisms, our definite knowledge of its precise manner of normal and abnormal function is meager in the extreme.

In this connection, it is justifiable to repeat the oft quoted, yet sometimes forgotten, maxim, that the proper basis for rational and effective therapeutics is clear physiologic knowledge. Any new light thrown upon the manner of function of a part of the body tends invariably to be reflected in an increase of effec-

tiveness as regards prevention, relief and cure of untoward conditions in relation to that anatomic territory.

This paper does not deal directly with the broader biologic questions of the precise mechanism of resistance, as such, in ciliated fields. Such questions are here noted in order to emphasize the need for more light in regard to the important functions of the ciliary system. We have limited ourselves to the investigation of certain specific physiologic mechanisms by means of which ciliated epithelium is regulated. Our paper deals with various hitherto unproved controls of the ciliated system. Experimental facts are submitted in detail to show that the ciliated epithelium is dominated by the same general types of organic regulatory devices as are the other kinds of visceral tissue of the body of the vertebrate organism. It is shown specifically that ciliated epithelium is dominated by neural, chemical and physical controls, which constitute a complex means of integrating the activity of this tissue in relation to the functional demands of the organism as a whole.

When the principles formulated in this paper had been determined by a study of amphibian cilia, our problem became one of finding out whether, and to what extent, the same principles hold for the cilia of higher vertebrates, including the human.

It may bring out more clearly the scope of the problem, if we compare the various physiologic methods by which the different kinds of visceral tissue are known to be controlled in the higher vertebrate with the hitherto assumed lack of similar controlling mechanisms for the visceral system represented by ciliated epithelium.

It is well known that the viscera of higher organisms possess the property of automaticity, that is, of being set in motion by intraviscerally generated excitations, and that the rate of activity so generated is subject to extrinsic regulation thru neural, chemical and physical influences; the neural control being mediated normally by the balance of opposing impulses of the sympathetic and parasympathetic groups of nerve fibers, and the chemical regulation being brought about by the secretions of specific endocrine glands and by certain products and by-products of metabolism. The physical types of control, where they exist, vary for the different viscera.

For example, we see this principle of intravisceral excitation and extravisceral control in the movement of the heart, which is set into activity by impulses arising in the sinoauricular

node, but is regulated by a balance of accelerating and inhibiting impulses thru the vagal and sympathetic divisions of the autonomic nervous system and thru the activity of hormonal substances elaborated in the body. Physical factors, such as the amount of filling and temperature, also play regulatory roles. The same principle has long been known to operate in the initiation and control of motion in the stomach and intestine, urinary bladder, and other visceral organs, which, tho generating intrinsically the impulses for their own activity, are also definitely regulated as to rate of movement, like the heart, by extrinsic neural and chemical influences as well as by certain physical agencies. In spite of the widespread recognition of these principles of neural chemical and physical regulation of movements arising from intraviscerally generated excitations, it is interesting that one important visceral system, the ciliated epithelial, has hitherto been held to possess only the power of automaticity without being subject in an orderly manner to such integrational regulatory influences.

Practically all writers upon the subject of ciliated epithelium deny that this type of visceral tissue is regulated by neural influence in higher organisms, altho it is well known that it does possess the property of automaticity, since it maintains its activity after excision from any possible general extrinsic nervous or hormonal connections. Typical of modern biologic and physiologic thought in this respect is the statement of Parker[2]: "The fact, however, that no one has ever been able to control ciliary activity thru nerves, and that all cases of ciliary coordination thus far brought forward can be explained on the basis of neuroid transmission, renders the belief in the nervous control of cilia extremely improbable. In fact, it may be stated at present that there is not the least ground for the assumption that true nervous activity is in any way involved in ordinary ciliary reactions. Yet, notwithstanding this independence of ciliated and nervous tissues, coordination in the activities of a ciliated field is one of its striking features."

It is interesting, that in spite of the physiologic importance which the ciliary apparatus possesses as part of various systems of higher organisms, it has been looked upon so long as being entirely independent of extrinsic organic regulation and control, maintaining as it were, complete anarchistic freedom to function at a locally determined automatic rate, without reference to the varied needs or exigencies of the organism as a

whole, in its manifold defensive reactions against noxious agencies in its environment.

Literature.

From the standpoint of structure, the cilium has been looked upon as the simplest form of motile tissue. Dellinger[3] says: "For pure contractile tissue, microscopically speaking, our simplest structure is the cilium. Here, with the protoplasm accessible for experiments with all kinds of reagents, if anywhere, we should find a key to the structure of contractile protoplasm."

On account of its importance, its extensive distribution thruout the different animal phyla, and its availability and apparent simplicity as a contractile form of protoplasm, ciliated epithelium has been widely studied. Since ciliary movement was discovered in 1683 by Anton de Heide, excellent accounts of it have been given by Engelmann,[4] by Frenzel[5] and many other authorities. Among these are Friedreich,[6] Marchi,[7] Eberth,[8] and Eimer.[9] Modern workers have done excellent research in this field. Grave and Schmitt[10] have described a beautiful mechanism, which they interpret to function as a means of interepithelial coordination. Saguchi[11] has given ciliary tissue extensive and exact embryologic and histologic study, and has discussed in considerable detail the function of its component structural elements. He does not, however, consider the larger question of the coordination of its activity with that of the higher organism as a whole, thru neural, chemical and physical regulatory agencies.

The function of ciliated epithelium has been studied and described extensively by Engelmann,[4] Verworn,[12] Merton,[13] Pütter,[14] and Metzner.[15] Interesting and exact studies of the effect of various physical and chemical agents upon ciliary activity have been made by Gray,[16] and others.

Agersborg[17] has recently noted nerve fibers extending from the basal ganglion of Melibe to the base of the epithelium of the foot. Merton,[13] working on the fresh water snail, has given evidence for nervous control of ciliated epithelium in this low form. Yet the weight of authority is entirely against the view that the action of ciliated epithelium is controlled by means of regulatory nerves.

We have performed many experiments which show conclusively that the activity of ciliated epithelium in the vertebrate (frog) is definitely subject to neural control, and to hormonal types of chemical control.[18] We have also shown that the

propulsive power of cilia is inhibited or paralyzed from lack of a normal surface film of moisture, which is, therefore, a highly important physical factor of control.[19] That ciliary activity is very sensitive to temperature, as already known, has been abundantly verified in our work.

Materials and Methods.

The present report is based upon observations made upon the activity of ciliated epithelium in the palate and pharynx of the frog, and upon the trachea, nasal cavities and paranasal sinuses of certain mammals as cat, dog and rabbit. The quantitative data, first to be described, were obtained from a study of the ciliated epithelium of the palate and pharynx of the frog. In the determination of these principles, the frog was used because it presents a large field of pharyngeal and oral epithelium, which is easy to study. The small size of the animal makes it convenient for experimental work in problems like the effects of chemicals upon its ciliated surfaces. Moreover, its nervous system, tho small, is organized so as to permit separate stimulation of the sympathetic and parasympathetic groups of nerve fibers which supply the ciliary regions studied.

Records of the ciliated speed rate were obtained as follows: We observed the time, in seconds, in which a field of ciliated epithelium propelled a particle of cellulose across the field of a binocular microscope. In each experiment, we shifted the particle repeatedly and noted and recorded its rate of speed for several separate journeys across the field. The average time, in seconds, of the journeys so obtained in the first part of each experiment was taken as an experimental norm with which to compare the average speed rate similarly obtained in the second part of the same experiment. In order to gain an unobstructed view of the ciliated field, the lower jaw was cut away, and the frog was held with pins upon its back upon a frog board.

In each experiment, after the average normal ciliary speed rate was ascertained by noting the time, in seconds, required by a particle to make several journeys across the field, its average speed was determined for a second series of journeys of the same particle across the same field while the nerve fibers, either sympathetic or parasympathetic to the pharyngeal and oral regions, were being electrically stimulated. Similar records were made before and after the administration of sympathomimetic and parasympathomimetic chemicals, which were injected into

the anterior lymph sac of the frog, and before and after evaporation, by means of gentle blasts of air blown from a rubber bulb, of the film of moisture from the surface of the mucosa.

The particle of cellulose used in the study of the cilia in the frog was obtained by taking a shred of loose, light paper and rolling it into globular shape. We found that the particle should be made very small. This particle was moistened before being placed upon the ciliated surface, since when dry it tended to become anchored upon the surface by absorption of moisture. Too large and heavy a particle tends to be unevenly propelled, and therefore its movement does not represent correctly the rate of ciliary activity. The paper particle was used by preference after trying several other materials in particulate form, such as lycopodium, carbon, etc. However, other types of particles, as will be noted later, have been employed in the study of the cilia of higher vertebrates.

Electric stimulation was obtained by means of an inductorium (Harvard Apparatus Co.) attached to a six volt electric cell. The stimulating current was of medium strength to the tongue. The stimulating electrode consisted of sharp steel needles, which were fixed about two millimeters apart, bent at the ends to hold the nerve, and attached by means of copper wire to the inductorium. For large frogs, we used the ordinary platinum electrodes employed in the physiologic laboratory (Harvard Apparatus Co.). We found that a plate holder (Harvard Apparatus Co.) attached by a double clamp to a ring stand makes a convenient electrode holder, the lead rod being easily moulded so as to bring and to hold the electrode in apposition with the nerve. In each experiment involving direct nerve stimulation, the required dissection was made and the electrode placed in position upon the nerve, sympathetic or parasympathetic, before the normal speed rate was established, as explained above.

Therefore, after the preliminary dissection had been done, and a speed norm had been established, the rate of ciliary movement *during* the stimulation of the nerve was found by simply closing the key of the primary circuit and performing the second part of the experiment without disturbing in any way the position of the ciliary field from which the normal speed rate had already been determined in the first part of the experiment. The result was, that in the experiments involving electrical stimulation, the ciliary speed rates before and after stimulation were obtained in each case by timing the rate of motion of the same

particle repeatedly across the same field, all the conditions being identical in both parts of the experiment, excepting that in the first part, the ciliary speed average, or experimental norm, was obtained *before* stimulation, and in the second part the speed average was obtained *during* stimulation. Both parts of each of the experiments with chemical and physical agents were also performed upon the same microscopic field.

In shifting the cellulose particle for repeated journeys, we found that care must be taken to prevent undue mechanical irritation of the subjacent epithelial cells.

On account of the secretion of mucus, which tended to interfere with the even rate of movement of the particle, we found it of advantage to wash the mucous membrane before both parts of each experiment. Since rough mechanical manipulation of ciliated epithelium impairs its propulsive power, the washing was done gently. The same amount of fluid (tap water, physiologic saline, or Ringer's solution) was used for washing the membrane in both parts of each experiment, and its application to the membrane was timed so as to precede both parts by equal intervals. The fluid was applied by a medicine dropper, which was also used in its removal, care being taken to draw the fluid off gently, without permitting the dropper to come into contact with the portion of the field directly under the microscope. The ciliated field was sloping when the animal's head was pinned to the cork covered frog board, the inclination of the field thus insuring equality of liquid drainage when the fluid was drawn off for each part of the experiment. The reason for these precautions was, of course, to keep all the experimental conditions parallel for the two parts of each experiment, excepting the particular conditions of neural, chemical and fluid control, the investigation of which formed the principal basis of our research.

Experimental.

Is the Rate of Ciliated Epithelium Accelerated as a Result of Sympathetic Nerve Stimulation?

The effect of electric stimulation of the sympathetic nerve trunk upon the rate of motion of ciliated epithelium is shown in Tables I and II, which represent the data of experiments one to nine, inclusive.

TABLE I.

Acceleration of the rate of motion of the ciliated epithelium of the mouth and pharynx of the frog thru electric stimulation of the sympathetic nerve trunk, as measured from the average rate of propulsion of a cellulose particle across the microscopic field before and during stimulation.

	Speed of particle in crossing microscopic field before stimulation		Speed of particle in crossing microscopic field during stimulation		Speed increase as result of stimulation
Experiment One	Journeys	Seconds	Journeys	Seconds	
	1	7	1	3	
	2	6	2	3	
	3	7	3	5	
	4	5	4	3	
	5	7			
	Average crossing time—6.4 seconds		Average crossing time—3.5 seconds		82.8%
Experiment Two	Journeys	Seconds	Journeys	Seconds	
	1	8	1	4	
	2	6	2	4	
	3	7	3	3	
	4	8	4	3	
	5	6	5	4	
	Average crossing time—7 seconds		Average crossing time—3.6 seconds		94.4%
Experiment Three	Journeys	Seconds	Journeys	Seconds	
	1	7	1	3	
	2	6	2	3	
	3	6	3	4	
	4	7	4	4	
	5	6	5	3	
	Average crossing time—6.4 seconds		Average crossing time—3.4 seconds		88%
Experiment Four	Journeys	Seconds	Journeys	Seconds	
	1	9	1	5	
	2	10	2	6	
	3	9	3	5	
	4	11	4	6	
	5	11	5	5	
	Average crossing time—10 seconds		Average crossing time—5.4 seconds		85%
Experiment Five	Journeys	Seconds	Journeys	Seconds	
	1	4	1	1	
	2	4	2	1	
	3	4	3	2	
	4	5	4	3	
	5	4	5	3	
	6	4	6	1	
	7	4	7	2	
	Average crossing time—4.14 seconds		Average crossing time—1.85 seconds		123.7%

In table I are five different experiments. Each experiment consists of two parts; the first part, which is the experimental speed norm, was taken before stimulation, and the second part, which shows the accelerating effect of sympathetic nerve impulses, was taken during stimulation. The first experiment is here described in detail as explanatory in general of the others, which will, therefore, be discussed in more general terms. In part one of the first experiment, therefore, five journeys of the cellulose particle across the microscopic field are each shown separately timed and recorded in seconds *before stimulation*. These journeys required 7, 6, 7, 5 and 7 seconds, respectively. The average of these five journeys of the particle across the field before stimulation is 6.4 seconds, which we adopted as a ciliary speed norm for the experiment.

In the second part of the same experiment, the average time, in seconds, of four journeys of the particle across the same microscopic field *during stimulation* required 3, 3, 5 and 3 seconds, respectively, making an average of 3.5 seconds.

When we compare the average speed of the particle before stimulation (6.4 seconds) with its average (3.5 seconds) during stimulation, we find a gain of 2.9 seconds. This represents an acceleration of ciliary activity of 82.8 per cent, which occurred as a result of stimulation of the sympathetic nerve in the region of the pharynx.

In the first part of experiment two, five journeys of the particle across the microscopic field before stimulation averaged 7 seconds. Five journeys of the particle during stimulation averaged 3.6 seconds. The second average is 3.4 seconds shorter than the first. This indicates, as in experiment one, an acceleration in the rate of ciliary motion, which in this case is 94.4 per cent.

In part one of the third experiment, five journeys of the particle across the microscopic field averaged 6.4 seconds. The average journey time of the same particle across the same field during stimulation was 3.4 seconds. The second average is here 3 seconds shorter than the first, and therefore represents an acceleration of 88 per cent as a result of sympathetic nerve stimulation.

In experiment four, performed on a cooler day, the time consumed by the particle in five journeys across the microscopic field before stimulation averaged 10 seconds. The average crossing time during stimulation was 5.4 seconds. The average here

is 4.6 seconds shorter than the first. The acceleration in ciliary motion was 85 per cent. The slower speed rate in both parts of this experiment was due to the fact that the experiment was performed on a cold day. The effect of cold in slowing ciliary activity has been shown by Kraft,[20] Gray[16] and others. However, the percentage rate of acceleration obtained from stimulation of the sympathetic is practically the same as in experiments performed in a warmer environment, when the ciliary speed rate is more rapid.

In experiment five, the average crossing time of five journeys of the cellulose particle before stimulation was 4.14 seconds. During stimulation, the average of four journeys was 1.85 sec-

TABLE II.

Acceleration of the rate of motion of the ciliated epithelium of the mouth and pharynx of the frog thru electric stimulation of the sympathetic nerve trunk, as determined from the average time of propulsion of a cellulose particle across the microscopic field before and during stimulation.

	Speed of particle in crossing microscopic field before stimulation	Speed of particle in crossing microscopic field during stimulation	Speed increase as result of stimulation
Experiment Six	Average 20 seconds	Average 10 seconds	100%
Experiment Seven	Average 20 seconds	Average 10 seconds	100%
Experiment Eight	Average 21 seconds	Average 10 seconds	110%
Experiment Nine	Average 30 seconds	Average 11 seconds	172%

Total average crossing time, experiments 1-9, 106%.

In this table each average represents several journeys across the microscopic field, as in experiments one to five in Table I. The individual journeys of the particle are omitted to save space. Note that the percentage of acceleration is higher from sympathetic stimulation when the initial speed of ciliary motion is slower on account of shock from experimental procedures. All the experiments in this table, as in Table I, show a marked acceleration from sympathetic stimulation.

onds, which was 2.29 seconds shorter than the first average, giving an acceleration of 123.7 per cent.

When we compare the speed norm of the cellulose particle across the microscopic field in 25 separate journeys before stimulation, as given in these five experiments, with its speed rate in the 23 journeys during stimulation, we find in every instance a marked acceleration as a result of the stimulation of the sympathetic. The average acceleration of all journeys so far considered during stimulation amounts to 94.98 per cent.

Further evidence as to the accelerating effect of stimulation of the sympathetic nerve trunk upon the rate of ciliary activity is given in experiments six, seven, eight and nine, table II. These results were obtained and recorded in the same manner as in the five experiments of table I, but to save space the average duration only of the journeys is given, the time required for the individual crossings having been omitted. In experiment six, performed in a cold laboratory, the average journey time of the particle was 20 seconds before stimulation and 10 seconds during stimulation. Here the stimulation of the sympathetic resulted in 100 per cent acceleration of ciliary activity.

The next three experiments, seven, eight and nine, were performed successively upon the same animal. In experiment seven, there was an acceleration of 100 per cent in the ciliary movement as a result of sympathetic stimulation, and in experiment eight an acceleration of 110 per cent. In experiment nine, the average rate of ciliary motion before stimulation was reduced from shock by procedure in experiments seven and eight. Stimulation of the sympathetic, however, caused an acceleration in this case of 172 per cent. The percentage acceleration from sympathetic stimulation was, therefore, much more in the shocked animal than in the fresher animal.

Partial Summary.

Experiments so far considered show that stimulation of the sympathetic trunk caused a marked acceleration in the rate of motion of the ciliated epithelium. With the exception of experiment nine, in which acceleration was relatively high in an animal whose normal rate was depressed from long continued experimental procedures, the rate of speed was in every case approximately doubled as a result of sympathetic stimulation. The average increase in the rate of ciliary motion in experiments one to nine, inclusive, was 106 per cent.

Is the Rate of Ciliary Activity Retarded as a Result of Stimulation of Parasympathetic Nerve Fibers?

Having thus determined that the ciliated epithelium studied is, like the heart, accelerated by stimulation of the sympathetic trunk, the question arose whether or not this epithelium was also subject to the control of the parasympathetic nerve fiber group.

A consideration of the dual plan of nerve regulation of the viscera of higher organisms, as the heart, urinary bladder, gastrointestinal tract, etc., suggested the probability of a parasympathetic nerve regulation for ciliated epithelium as well as a sympathetic. Since the action of the sympathetic nerve supply is accelerating to the ciliated epithelium, we naturally postulated that the parasympathetic nerves should have a depressant effect.

The determination of this latter point we found technically much more difficult than the former. When we stimulated the nerves to the oral and pharyngeal regions of the frog, after their emergence thru the cranial foramina, the results were inconclusive: we found neither a definite acceleration nor retardation. We postulated that the indifferent results were due to the fact, that at this distance from the brain, the nerves to the region studied probably contained both sympathetic and parasympathetic fibers, which should tend to neutralize each other. Opening the cranial cavity and exposing the brain, we stimulated the cranial nerves bearing parasympathetic fibers, that is, the VII, IX, X and XI, close to the points of their attachments to the medulla oblongata. After several experiments, we found that an easier method of stimulating these fibers is by piercing the medulla with the needles of the electrode and leaving them in this position. Before stimulation, we isolated the medulla oblongata from the spinal cord and the midbrain by complete transections, in order to prevent reflexes extending to other body levels. We destroyed the cerebral hemispheres, midbrain and spinal cord without disturbing the medulla. Then we found and recorded the effects of stimulation of the parasympathetic nerve fibers upon ciliary activity by the same methods as those used in the experiments upon the sympathetic, as already described.

The effect of stimulation of the parasympathetic nerves upon the rate of activity of ciliated epithelium is shown in experiments ten and eleven, table III. In experiment ten, the average of four journeys of a particle across the microscopic field was nine seconds before stimulation, and 16 seconds during stimu-

TABLE III.

Retardation of the rate of motion of the ciliated epithelium of the mouth and pharynx of the frog thru electric stimulation of the parasympathetic nerves, as determined from the average time of propulsion of a cellulose particle across the microscopic field before and during stimulation.

	Speed of particle in crossing microscopic field before stimulation		Speed of particle in crossing microscopic field during stimulation		Decrease in speed as a result of stimulation
Experiment Ten	Journeys	Seconds	Journeys	Seconds	
	1	9	1	12	
	2	10	2	21	
	3	8	3	18	
	4	9	4	13	
	Average crossing time—9 seconds		Average crossing time—16 seconds		77.7%
Experiment Eleven	Journeys	Seconds	Journeys	Seconds	
	1	12	1	21	
	2	10	2	27	
	3	11	3	35	
	4	11	4	18	
	5	11	5	23	
	6	12	6	19	
	7	11	7	20	
	8	10	8	25	
	9	10	9	20	
	10	10	10	20	
	11	10	11	19	
	Average crossing time—10.7 seconds		Average crossing time—22.45 seconds		109.8%

lation. The second average is 7 seconds slower than the first, which represents a retardation of 77.7 per cent.

In experiment eleven, upon another animal, the average of eleven journeys of the particle before stimulation was 10.7 seconds. During stimulation there was a marked slowing of ciliary activity, the average journey time now being 22.45 seconds. This represents a retardation of 109.8 per cent.

Experiments eight and nine are typical of the results obtained in many similar experiments. In all of these experiments which involved stimulation of the parasympathetic nerve fibers, we obtained uniformly a marked retardation of the rate of motion of the ciliated epithelium in the region studied.

Partial Summary.

The experiments so far considered show that stimulation of the sympathetic nerves of the vertebrate (frog) accelerates markedly ciliary activity in the region studied, and stimulation of the parasympathetic just as markedly inhibits this movement.

The Possible Influence of the Vascular Factor Ruled Out.

The question arose whether vascular changes might be a factor involved in the acceleration or depression of ciliary activity which followed upon nerve stimulation. Veach[21] has submitted evidence which he interprets to the effect, that the relaxation of the stomach (cat) which follows stimulation of the great splanchnic nerve is not a primary response of visceral muscle to the neural stimulus, but is secondary to the limitation of blood supply which results from vasoconstriction. Lommel[22] found that anemia slowed ciliary movements. It was important to determine whether the vascular factor might not also be causatively related to the modification of ciliary activity we were obtaining. The incidental evidence that occurred during our experiments did not support this hypothesis. There were no gross vascular changes recognizable during nerve stimulation, such as would be indicated by marked blanching or flushing. Also, extensive bleeding did not prevent the accelerating or retarding effects of nerve stimulation, if the experiments were not too long delayed. Cutting of both vagi peripherally to the pharynx did not alter recognizably the usual rate of ciliary retardation when the parasympathetic nerves were stimulated, showing that retarded ciliary activity was not the result of ischemia due to cardiac inhibition. This indirect evidence, tho suggestive, was not considered conclusive in ruling out the vascular factor.

That acceleration during sympathetic stimulation, however, was not an indirect result of vascular changes is conclusively shown by experiment twelve, table IV, in which the heart was excised before the ciliary speed rate was taken in both parts of the experiment. After cardiac excision, the average rate of six journeys of the cellulose particle across the field of the microscope before stimulation was 10.5 seconds. The average rate of the same particle for six journeys across the same field during stimulation was 5.16 seconds, an increase of 103 per cent.

TABLE IV.

Acceleration of the rate of motion of the ciliated epithelium of the mouth and pharynx of the frog thru electric stimulation of the sympathetic, as determined from the average time of ciliary propulsion of a cellulose particle across the microscopic field, after excision of the heart.

	Crossing time of particle before stimulation of the sympathetic, after heart excision		Crossing time of particle during sympathetic stimulation, after heart excision.		Acceleration of cilia from sympathetic stimulation
Experiment Twelve	Journeys	Seconds	Journeys	Seconds	
	1	10	1	5	
	2	10	2	5	
	3	11	3	5	
	4	11	4	4	
	5	11	5	7	
	6	10	6	5	
	Average 10.5 sec.		Average 5.16 sec.		103%

Retardation of the rate of motion of the ciliated epithelium of the mouth and pharynx of the frog thru electric stimulation of the parasympathetic, as determined by the average time of propulsion of a cellulose particle across the microscopic field, after excision of the heart.

	Crossing time of particle before stimulation of the parasympathetic before heart excision		Crossing time of particle during stimulation of the parasympathetic after heart excision		Retardation from parasympathetic stimulation, after heart excision
Experiment Thirteen	Journeys	Seconds	Journeys	Seconds	
	1	12	1	28	
	2	10	2	20	
	3	13	3	21	
	4	14	4	18	
	5	11	5	24	
	6	12	6	26	
	7	10	7	19	
	Average 11.7 sec.		Average 22 28 sec.		90.4%

That the retardation resulting from parasympathetic stimulation was not an indirect result of vasomotor changes is also shown in experiment thirteen, table IV. The average rate of

several journeys of the particle after heart excision and before stimulation was 11.7 seconds. The average rate of seven journeys of the particle during parasympathetic nerve stimulation was 22.28 seconds. This represents a retardation of 90.4 per cent.

These two experiments show conclusively, it seems, that the variation of ciliary activity which is caused by stimulation of the sympathetic and parasympathetic nerves is not due to changes in the blood supply, since the rate of acceleration and retardation in both experiments was not materially influenced by elimination of the blood flow thru heart excision.

The Effect of Sympathomimetic and Parasympathomimetic Chemical Substances.

Since the studies detailed above show that ciliated epithelium is, like other contractile tissues which occur in various visceral systems of higher organisms, accelerated and inhibited by the activity of the sympathetic and parasympathetic components of the automatic nervous system, it seemed that cilia should, like these splanchnic mechanisms, be subject also to acceleration and depression by action of sympathomimetic and parasympathomimetic hormones and chemicals of similar pharmacologic actions.

In order to throw light upon this point, we studied the effects of several sympathomimetic and parasympathomimetic drugs upon ciliary activity. The hormone, epinephrin, and the vegetable drug, ephedrin, were the sympathomimetic substances employed. Pilocarpin and atropin were used as typical parasympathomimetic chemical agents.

Is Ciliary Activity Accelerated Thru the Action of Sympathomimetic Chemical Substances?

The effect of epinephrin is given in experiment fourteen, table V. Since this hormone is known to stimulate the sympathetic at its myoneural junction, and since our experiments show that sympathetic stimulation accelerates ciliated epithelium, we naturally expected an acceleration in the rate of ciliary movement by the use of epinephrin (adrenalin, Parke Davis & Co.). Our anticipation was verified, as shown in experiments fourteen and fifteen. These experiments require explanation somewhat in detail. The average time of eleven journeys of the particle

TABLE V.

Acceleration of the rate of motion of the ciliated epithelium of the mouth and pharynx of the frog by the action of epinephrin, as determined by the average time of propulsion of a cellulose particle across the microscopic field before and after administration of the hormone.

	Speed of particle in crossing microscopic field before administration of epinephrin.		Speed of particle in crossing microscopic field after administration of epinephrin		Increase of speed thru action of epinephrin
	Journeys	Seconds	Journeys	Seconds	
Experiment Fourteen	1	15	1	13	
	2	14	2	10	
	3	12	3	10	
	4	10	4	9	
	5	10	5	7	
	6	11	6	9	
	7	10	7	6	
	8	10	8	6	
	9	9	9	6	
	10	10	10	6	
	11	10	11	4	
	Average crossing time—11 seconds		12	9	
			13	10	
			14	11	
			15	11	
			16	10	
			17	11	
			Average crossing time of journeys 5-11—6.28 sec.		75%
Experiment Fifteen	Journeys	Seconds	Journeys	Seconds	
	1	18	1	15	
	2	17	2	12	
	3	18	3	13	
	4	17	4	12	
	5	21	5	10	
	Average crossing time—18.2 seconds		6	11	
			7	11	
			8	13	
			9	9	
			10	8	
			11	9	
			12	9	
			13	9	
			14	10	
			15	10	
			Average crossing time of journeys 9-13—8.8 sec.		106.8%

across the microscopic field before administration of .1 mg. of epinephrin was 11 seconds. The average rate of ciliary motion after the drug was injected and had time to bring about its characteristic action is shown in journeys 5 to 11 of the second part of the experiment. Here as we had anticipated, the effect was one of acceleration.

Owing to the fleeting action of the drug, the acceleration soon passed off, and in journeys 13 to 17 in this part of the experiment, the epithelial action settled back to its rate before administration of the hormone. The acceleration during the greatest action of the hormone (journeys 5 to 11 inclusive) was 75 per cent.

In experiment fifteen, the accelerating effect of epinephrin is also evident, the average acceleration of fifteen journeys of the particle being 70 per cent. As in experiment fourteen, the hormone shows its greatest activity during a few journeys; in this case, those from 9 to 13 are most rapid, showing an acceleration during these journeys of 106.8 per cent.

Since epinephrin is a sympathomimetic substance, the ciliary acceleration obtained by its action supports the conclusion already

TABLE VI.

Acceleration of the action of ciliated epithelium of the mouth and pharynx of the frog by the action of ephedrin sulphat, as measured by the average time of propulsion of a cellulose particle across the microscopic field before and after the administration of the compound.

	Speed of particle in crossing microscopic field before the administration of ephedrin sulphat.		Speed of particle in crossing microscopic field after the administration of ephedrin sulphat		Increase ciliary motion by action of ephedrin sulphat
Experiment Sixteen	Journeys	Seconds	Journeys	Seconds	
	1	11	1	8	
	2	10	2	8	
	3	14	3	4	
	4	14	4	7	
	5	11	5	7	
	6	12	6	6	
	7	9	7	7	
	8	13	8	6	
	9	13	9	7	
	10	14	10	9	
	Average crossing time—12.1 seconds		Average crossing time—6.9 seconds		75.3%

arrived at by the method of direct stimulation of the sympathetic nerves, namely, that the rate of movement of ciliated epithelium is accelerated thru the activity of the sympathetic nervous system.

The effect of another sympathomimetic chemical substance, ephedrin sulphat, is given in experiment sixteen, table VI. This interesting old Chinese drug is now known to resemble epinephrin considerably, both chemically and pharmacologically, tho its pharmacodynamic efforts are much longer in duration. The average rate of speed of the ciliated epithelium before the injection of ephedrin was 12.1 seconds. The average rate after its administration was 6.9 seconds. As with epinephrin, there was an acceleration in the rate of motion of the ciliated epithelium, which amounts in this case to 75.3 per cent.

Is Ciliary Activity Modified Thru the Action of Parasympathomimetic Chemical Agents?

Having determined that the sympathomimetic drugs, epinephrin and ephedrin, cause an acceleration of ciliary activity similar to that obtained by direct stimulation of the sympathetic, we then tested the effect upon the rate of ciliary action of the parasympathomimetic drugs, pilocarpin and atropin. The effects of pilocarpin are given in table VII, experiments seventeen and eighteen.

Before the administration of pilocarpin, the average rate of ciliary activity in experiment seventeen was 12.1 seconds. After .2 mg. of the drug was injected, ten more journeys of the particle were timed, when the ciliary speed average was found to be 22.6 seconds. The difference between the two averages is 10.5 seconds, which indicates a retardation amounting to 86.7 per cent.

We obtained similar results in the next experiment, eighteen, performed upon a different animal. Before administration of .2 mg. of pilocarpin, the average journey time of the cellulose particle across the microscopic field was 10.1 seconds. After injection, the ciliary motion was retarded to an average of 21.7 seconds for the journeys. The second average is 11.6 seconds slower than the first, which gives a retardation of 114.8 per cent.

The parasympathomimetic drug, pilocarpin, therefore inhibits the rate of action of the ciliated epithelium.

The effect of atropin upon the rate of ciliary motion is shown in experiments nineteen and twenty, table VIII. The results here are not so simple as in the preceding experiments, since to a considerable extent atropin gives effects which are antagon-

istic to each other in their influence upon ciliary propulsion. On account of the well known blocking action of atropin at the parasympathetic terminals (myoneural junction) we naturally expected a ciliary acceleration as a result of unopposed sympathetic action. We found, however, that its first effect may be to slow ciliary propulsion considerably. The principal cause of slowing seemed to be the adhesive and drying effect of the drug upon the mucosa by modification of gland action, tho early preparalytic irritative action of the drug may have also been a factor in the initiation of ciliary retardation. When the mucosa was

TABLE VII.

Retardation of the rate of motion of ciliated epithelium of the mouth and pharynx of the frog by the action of pilocarpin, as determined by the average rate of propulsion of a cellulose particle across the microscopic field before and after administration of this compound.

	Speed of particle in crossing microscopic field before the administration of pilocarpin		Speed of particle in crossing microscopic field after the administration of pilocarpin		Decrease of speed thru action of pilocarpin
Experiment Seventeen	Journeys	Seconds	Journeys	Seconds	
	1	11	1	30	
	2	10	2	25	
	3	14	3	32	
	4	14	4	32	
	5	11	5	17	
	6	12	6	14	
	7	9	7	17	
	8	13	8	18	
	9	13	9	26	
	10	14	10	15	
	Average crossing time—12.1 seconds		Average crossing time—22.6 seconds		86.7%
Experiment Eighteen	Journeys	Seconds	Journeys	Seconds	
	1	11	1	20	
	2	11	2	21	
	3	11	3	26	
	4	9	4	24	
	5	10	5	21	
	6	10	6	29	
	7	10	7	20	
	8	9	8	20	
	9	10	9	18	
	10	10	10	18	
	Average crossing time—10.1 seconds		Average crossing time—21.7 seconds		114.8%

washed, however, the activity of the cilia was definitely accelerated, as shown in the table.

Atropin, therefore, has an accelerating effect upon ciliary propulsion when the membrane is kept sufficiently moist and free from a sticky surface covering which retards or prevents the pro-

TABLE VIII.

The effect of .2 mg. of atropin upon the rate of ciliary activity, as measured by the average rate of propulsion of a cellulose particle across the microscopic field before and after the administration of the drug.

	The normal rate of ciliary propulsion before the injection of atropin.		The immediate effect of .2 mg. of atropin upon the rate of ciliary propulsion.		The rate of propulsion after washing the membrane, following atropin injection.		Acceleration
Experiment Nineteen	Journeys	Seconds	Journeys	Seconds	Journeys	Seconds	
	1	22	1	40	1	13	
	2	29	2	28	2	10	
	3	20	3	31	3	10	
	4	18	4	18	4	11	
	5	24	5	23	5	13	
	6	27					
	Average crossing time—23 sec.		Average crossing time—28 sec.		Average crossing time—11.4 sec.		100%

When atropin is first injected, the membrane becomes so dry and sticky that the ciliary propulsion is somewhat slowed. When the membrane is washed, the ciliary propulsion was markedly accelerated.

	The normal rate of ciliary propulsion.		Speed of particle in crossing microscopic field 6 minutes after injection of .3 mg. of atropin.		Rate of propulsion after washing the mucosa following atropin injection.		Acceleration
Experiment Twenty	Journeys	Seconds	Journeys	Seconds	Journeys	Seconds	
	1	16	1	26	1	17	
	2	17	2	16	2	15	
	3	17	3	19	3	11	
	4	18	4	15	4	10	
	5	16	5	15	5	12	
	6	19	6	10	6	12	
					7	10	
					8	9	
					9	8	
					10	8	
					11	9	
					12	9	
					13	7	
					14	7	
	Average crossing time—17.1 sec.		Average crossing time—16.8 sec.		Average crossing time—9.27 sec.		84%

pulsive movement of the particle. Experiment twenty shows, also, the accelerating effect of atropin upon the rate of ciliary propulsion, before and after the mucosa had been cleaned and moistened.

Ciliary inhibition and paralysis due to dehydration and cooling were as easy to demonstrate for the higher vertebrate as for amphibian. The question of neural and chemical control of cilia in the higher vertebrates is now under investigation by the senior author. The results so far obtained support the principle of neural and chemical regulation of cilia in the higher animal forms. This important point requires considerable research, and is being checked up by several methods before final conclusions will be drawn.

Partial Summary.

These experiments show clearly, then, that (1) the rate of ciliary activity is markedly accelerated by direct stimulation of the sympathetic nerves, and by the action of the sympathomimetic drugs, epinephrin and ephedrin sulphat; that (2) it is also markedly delayed, or inhibited, by direct stimulation of the parasympathetic nerves, and by the action of the parasympathomimetic drug, pilocarpin; and that (3) atropin has mixed effects upon ciliary activity, retarding its propulsion at first, mainly by drying and thickening the surface moisture despite a faster movement of the cilia, but accelerating propulsion after the mucosa is cleaned and moistened.

Stated in more general terms, we may say that ciliary epithelium, long known to possess the property of automaticity, like the various organs of the body which are composed of visceral tissue, is also in the region studied subject like these organs generally to extrinsic regulation by the dual influence of the sympathetic and parasympathetic, and by the action of sympathomimetic and parasympathomimetic chemical substances.

The Effect of Drying Upon the Rate of Ciliary Propulsion.

The rate of ciliary propulsion as noted by Gray[16] and others is highly sensitive to changes in temperature. This point was abundantly confirmed in our work. When the atmosphere is cold, the journey of the particle may require several minutes. We have not incorporated such slow records in this paper. On hot days, the particle crossed the field very quickly, the individ-

ual journeys, especially during sympathetic stimulation, sometimes requiring only one second. In fact, the ciliary movement was then so swift that it was difficult to time correctly its rate of propulsion. The markedly accelerating effect of heat is shown in experiment five, which was performed on a fairly hot July day (room temperature 87.9° F.)

We find that another physical factor, namely moisture, has an even more profound effect than temperature changes upon the rate of ciliary propulsion. The relation between ciliary motion and the amount of moisture present upon the membrane is shown in experiments twenty-one, table IX, and twenty-two, table X.

TABLE IX.

The effect of drying of ciliated epithelia upon their rate of propulsion.

	Speed of particle in crossing microscopic field before drying.	Mucous membrane dried by air pump until sheen disappeared.	Speed of particle after cleaning with water the sticky mucous membrane.
Experiment Twenty-one	Journeys Seconds 1 14 2 14 3 13 4 14 5 15	No motion whatsoever for fifteen minutes.	Journeys Seconds 1 23 2 27 3 23 4 30 5 40 6 32
	Average crossing time—14 sec.		Average crossing time—30.8 sec.

Note (1), that the normal rate of ciliary propulsion is here 14 seconds;
(2), that there was an entire cessation of ciliary propulsion after the moisture upon the mucous membrane was evaporated by draughts of air; and
(3), that there was a resumption of propulsive power after moistening but with some impairment as shown by a speed rate slower than normal.

In experiment twenty-one, the average rate of propulsion of the particle across the microscopic field before evaporation of the moisture was 14 seconds. After evaporation of the surface moisture by gentle air draughts from a rubber bulb, there was no propulsive motion whatsoever for fifteen minutes. But when the viscous film was washed away, propulsion was again resumed over the moistened mucosa, but at a considerably slower rate.

TABLE X.

The effect of drying upon the rate of propulsive action of ciliated epithelium in the palate and pharynx of the frog.
(Experiments 22 to 22f performed successively upon the same animal).

	Speed of particle in clossing microscopic field before drying.		The retarding effect of drying the mucous membrane.
Experiment Twenty-two	Journeys / Seconds 1 — 16 2 — 12 3 — 14 4 — 17 5 — 15 6 — 18 7 — 17 Average crossing time—15.28 Seconds	Experiment 22a	The mucous membrane was partially dried by gentle draughts of air for 5 minutes. Particle at standstill for 5 minutes, then begins to move very slowly as membrane becomes moistured by secretion. Acquires more speed as moisture returns. The particle crosses the microscopic field in 15.5 minutes. Was 5 minutes starting and 10 minutes crossing.
	Propulsive action resumed when moisture is again added.		Moist mucosa not washed.
Experiment 22b	Journeys / Seconds 1 — 20 2 — 20 3 — 21 4 — 17 5 — 20 Average—19.6 second	Experiment 22c	Journeys / Seconds 1 — 24 2 — 17 3 — 15 4 — 19 5 — 19 Average—18.8 seconds
	One drop of moisture added to mucous membrane.		The effect upon the rate of ciliary propulsion of a second drying.
Experiment 22d	Journeys / Seconds 1 — 22 2 — 21 3 — 22 4 — 29 5 — 16 Average—22 seconds	Experiment 22e	Dried mucous membrane five minutes by means of air draughts. Placed a little ball of cotton just back of the upper jaw (frog lying on back) and upon free cut edges of base of lower jaw to prevent moisture from seeping into the ciliated field. Mucous membrane dry and brown over eye prominences, but a little moist and very sticky in mid furrow where particle was located. Particle goes ½ way across the field in ½ hour.

	Return of propulsive power upon moistening mucous membrane after second drying.		
Experiment 22f	Journeys	Seconds	
	1	19	
	2	31	
	3	33	
	4	27	
	5	21	
	Average—28.2 sec.		

Note: (1) that in the first part of experiment Twenty-two, when the mucous membrane was moist, the ciliated epithelium propelled the particle across the microscopic field in about 15 seconds;

(2) that partially drying the mucosa (22a) retarded the propulsive rate to over 15 minutes, which is sixty times as slow as before drying. No movement in first half of this period. Propulsion gradually returned as moisture was secreted;

(3) that when the mucous membrane was moistened again (22b) its speed of propulsion was increased to 19.6 seconds, or nearly that of the experimental normal;

(4) that when the mucous membrane was again dried by air currents (22e) the particle crossed the field one-half way in one-half hour. In the first part of this period there was no propulsive movement, but as the cilia obtained a little moisture from subjacent glands, the particle began to move very slowly. In experiments where the mucosa is kept dry, propulsive movement is entirely lacking;

(5) that there is a return of propulsive power (22f) when the mucosa is again moistened.

The same paralyzing effect upon the cilia by fluid loss is shown in experiment twenty-two. We see here again a paralysis of ciliary activity by drying, and a resumption of propulsive power when moisture is again added upon the mucosa. In this experiment, paralysis is brought about twice by evaporation of surface moisture, and ciliary function is again restored in each case by the addition of moisture.

The utter dependence of ciliated epithelium for propulsive function upon a surface film of moisture is clearly seen by these two experiments, which are typical of many similar experiments. Generally, as the mucosa loses moisture, a ciliary paralysis always develops pari passu with the fluid loss. When moisture is added the ciliary propulsion is restored.

However, the restoration of propulsive function after dehydration paralysis depends entirely upon the extent of drying, as shown in experiment twenty-three, table XI. When the membrane loses fluid until no moisture can be seen at the base of the cilia by means of the microscope, and the surface film loses en-

TABLE XI.

The effect of drying upon the rate of propulsive action of ciliated epithelium in the mouth and pharynx of the frog.

	Speed of particle in crossing microscopic field before drying.		The paralyzing effect of drying the mucosa.
Experiment Twenty-three	Journeys	Seconds	Membrane dried by gentle draughts of air till no longer adhesive. Results: No propulsive action whatever for thirty minutes, and individual cilia all over palate at a stand-still as seen thru microscope. Mucosa then kept moist for a half hour. Results: No return of propulsive power. Completely dried cilia showed no return of motion as seen thru the microscope. However, at periphery and front of palate, where drying was less complete, a few cilia could be seen beating in a few areas, but their action was insufficient to cause propulsion of the particle.
	1	11	
	2	13	
	3	11	
	4	12	
	5	10	
	6	11	
	7	11	
	Average—11.28 sec.		

tirely its adhesiveness and is kept dry for some time, then, adding moisture does not restore ciliary propulsive function at all. The cilia remain utterly paralyzed during the time devoted to the experiment, that is one to two hours.

When we discovered that the ciliary system loses propulsive power by moisture loss, the question arose whether the individual cilia themselves might still be undergoing movement tho unable to propel particulate material on account of increased adhesiveness and of the loss of a moisture film which, when present, lightens the weight of particles through hydrostatic buoyancy. It seemed possible that the vibratile power of the individual cilia might still be retained even after cessation of its propulsive ability thru drying.

This point was easily determined by microscopic study of the activity of the individual cilia during varying degrees of moisture evaporation. The whipping movements of the individual cilia were always seen to become slower and slower as the surface moisture diminished, and always came to a complete standstill when the film of moisture bathing the cilia was entirely evaporated.

The propulsive action, however, was practically lost before all the cilia had entirely stopped their vibratile movements. This was due to the adhesiveness of the surface film as the moisture

evaporated. As long as there was moisture enough upon the ciliated membrance to make it adhesive, some movements of the individual cilia could be seen, tho they were unable to propel the particle noticeably on account of the adhesiveness. Generally, areas of cilia at lower levels upon the uneven surface of the mucosa retained vibratile action after the higher lying, dryer cilia had entirely stopped vibrating. The senior author has shown, as will be noted later, that the cilia of respiratory surfaces of higher vertebrates, as cat and rabbit, react to temperature and moisture in a similar manner to that here described for the frog.

After completion of our experiments we noticed that Gray[16], working upon lower biologic forms, has shown that the activity of cilia is stopped by hypertonic salt solution, and he assumed that the cessation of action resulted from moisture lack, tho other possible chemical and physical factors incident to salt action were not ruled out. Our direct experiments prove conclusively, it seems, that moisture loss inevitably causes ciliary paralysis.

Hence, in addition to the neural and hormonal controls of ciliary propulsion, we may add the important physical control of moisture. These various controls have not hitherto been demonstrated in the physiology of the ciliary system. The effect of cold, acids, organic and inorganic, and various types of chemicals have been comprehensively described by Gray[16] and others. In fact, Gray[16] stated, on the basis of such studies, that the reactions of ciliated epithelium in lower forms to certain ions is like that of the heart. It is interesting that ciliated epithelium also in the mouth and pharynx of the frog is accelerated and inhibited, as shown above, by exactly the same influences, neural and chemical, as is the heart of the vertebrate.

Discussion.

The idea that gave rise to this research was suggested by a comparison of the physiologic devices, neural, chemical and physical, which are recognized generally as regulating the rate of automatically produced activity of the various viscera with the assumed lack of these same regulatory mechanisms for the coordination of the movements, similarly generated, of ciliated epithelium. The question arose: Since the motor activity of the various visceral systems, circulatory, digestive, excretory, etc., are delicately and efficiently coordinated by neural and chemical and physical mechanisms of regulation, why should not the activity of the ciliated epithelial mechanism stand subject to sim-

ilar means of organic coordination and integration? Why should not the federal demands of the entire organism as a composite biologic unit, growing out of the exigencies of its struggle to protect itself in a hostile environment and to dominate and utilize external and internal sources of material and energy, be imposed upon the visceral ciliated mechanism as imperatively as upon any other visceral system? Indeed, the demand for regulation seemed more imperative for the ciliary than for the other visceral systems, when it was recalled that ciliated epithelium of the higher vertebrate bears the direct brunt of the indispensable function of protecting the organism against vast numbers of microscopic enemies and various kinds of noxious materials more or less constantly present in the inspired air. Cilia also cause outward currents in the female genital system, which is in more or less free communication with the peritoneal cavity, the infection of which is so serious. Theoretically, it seems that freedom of the ciliary apparatus from the types of control laid upon the other visceral systems should contravene to that extent the basic law of organic unity imposed upon all biologic beings, however simple or complex. Practically, such lack of efficient coordination should leave the body less able to cope with serious dangers more or less continuously present in its habitat.

The data here submitted proves that the activity of ciliated epithelium in the regions studied possesses physiologic mechanisms of coordination, neural, chemical and physical, similar to those possessed by the dynamic tissue elements of other visceral systems. The highly important question arises: Is the ciliated epithelium of higher vertebrates, including man, subject to regulation by similar physiologic agencies? Is it subject to acceleration and retardation thru the activity of the visceral nervous system, and thru the actions of hormones and other types of chemicals, as well as by such physical factors as fluid and temperature?

The senior author is engaged in research with the view of determining whether fundamental principles of ciliary control, such as those described above for the amphibian, apply in general to the activities of the cilia of the higher vertebrates, including man. Some of the data and conclusions resulting from this work will be submitted at the October meeting of the American Academy of Ophthalmolgy and Oto-laryngology.

The potential clinical significance of the principles here enumerated, as determined from the study of the lower vertebrate,

depends, of course, upon the extent to which the ciliated epithelium of the higher vertebrate is dominated by similar physiologic mechanisms.

It may be said, by way of anticipation, that the work of the senior author points directly to the existence of neural, chemical and physical control mechanisms for the regulation of the rate of ciliary activity in the higher vertebrates, also.

It was easy to show that drying has the same paralytic effect upon the cilia of the respiratory systems of animals like the cat and rabbit as upon the amphibian ciliary apparatus. Experiment 24 shows the effect of drying. Even a slight amount

TABLE XII.

The effect of drying upon the rate of propulsion of ciliated epithelium in the trachea of the mammal (dog). The animal was decerebrated and 3½ hrs. time was permitted to elapse after decerebration before the experiment was begun. The paralyzing effect of the anesthesia upon the cilia had worn off. (Animal kept warm by heat of electric light bulbs, room temperature 79° F.)

	Rate of ciliary action before drying.		Rate of ciliary action after the mucosa of the open trachea was dried by gentle blasts of air for one minute.	Rate of ciliary action after the mucosa was remoistened with Ringer's solution.	
Experiment Twenty-four	Journeys	Seconds	Particle traveled ¼ to ⅓ across the field in 15 minutes. At that rate it would cross the field in 45 minutes to 1 hour. Its rate was therefore, 150 to 180 times as slow as before drying.	Journeys	Seconds
	1	18		1	22
	2	14			
	3	21.5		Further drying stopped particle completely.	
	4	24			
	5	23			
	Average crossing time—20.1 sec.				

of evaporation of moisture from the respiratory surface, such as the lining of the trachea, is sensitively reflected in a retardation of ciliary propulsion. Such ciliary slowing generally proceeds pari passu with the evaporation up to a certain point, when complete stoppage of ciliary propulsion supervenes.

It was interesting to note that currents of dry air seem to inhibit ciliary action much more quickly than currents of air having a high humidity, when the temperature is approximately the same. This was to be expected, since air currents of low relative humidity have a correspondingly greater drying effect.

Excess moisture upon ciliated surfaces also modifies ciliary propulsion. A ciliary surface normally propels its surface film in

a given direction. A greater amount of fluid, however, causes surface gravity currents, which flow to lower levels, irrespective of the direction of flow of the underlying fluid which is being propelled by the cilia.

Cooling of the ciliary surfaces of higher vertebrates is another important physical factor which retards the ciliary function in a similar manner to its action upon this tissue in lower forms. A slight cooling of the surface of the trachea of the cat or rabbit, for instance, causes a minimal slowing of ciliary motion, further cooling causes a further slowing, until, at a certain low temperature, ciliary activity entirely ceases. Cilia, here, thus behave like other types of contractile tissue, e. g., the different forms of muscle, which are well known to be retarded and finally entirely inhibited by cold.

TABLE XIII.

Retardation of the rate of activity of ciliated epithelium of the excised trachea of the dog as a result of cooling. Temperature of conditioning room, 98.5° F. Cooling done by means of flushing with saline in wash bottle connected by tube to trough in which the trachea was pinned open upon a paraffin covered incline.

	Rate of propulsion of ciliated epithelium at room temperature, 98.5° F.		Rate of propulsion of ciliated epithelium after flushing cilia with Ringer's solution at 89° F.		Rate of propulsion of ciliated epithelium after flushing with Ringer's solution at 63° F.	
Experiment Twenty-five	Journeys	Seconds	Journeys	Seconds	Journeys	Seconds
	1	5.4	1	15.0	1	62
	2	5.4	2	12.2	Washed with Ringer's solution at 58° F. For several minutes there was no visible motion. But as the trachea was gradually warmed from the hot room temperature ciliary activity returned but very slowly.	
	3	6.0	3	11.2		
	4	6.8	4	9.0		
	5	6.6	5	10.0		
	6	6.2	6	9.4		
	7	6.4	7	7.6		
	Average Crossing Time—6.1 Sec.		Average Crossing Time—10.6 Sec.			

The importance of the controlling mechanisms of ciliated epithelium from the point of view of normal and abnormal physiology will not be discussed until the research now under way upon this problem is completed.

I desire to thank Professor W. H. Howell, Director of the School of Hygiene and Public Health of Johns Hopkins University for many kind suggestions in regard to my work in his laboratory upon the cilia of higher vertebrates. With the excep-

tion of tables XII and XIII, however, the data of the research done there will be omitted here, and given in a later publication. I wish to thank Dean von W. Schulte for his kindness in loaning me some of the equipment from the department of anatomy which was used in this research. I wish also to thank Dr. Herbert F. Gerald, Professor of Pharmacology, for his advice as to dosage of the chemicals used in the experiments upon the amphibia.

General Summary.

1. Ciliated epithelium, as demonstrated in the amphibian, is subject to acceleration thru stimulation of the sympathetic, and to retardation thru stimulation of parasympathetic nerves.

2. It is also accelerated by the sympathomimetic chemicals, epinephrin (adrenalin) and ephedrin, and retarded by the parasympathomimetic chemical, pilocarpin. Atropin gives mixed effects, accelerating the cilia apparently by parasympathetic blocking, but also slowing propulsion by drying and thickening the moisture upon the cilia.

3. Ciliary activity is absolutely dependent for function upon a surface film of moisture, being retarded and then paralyzed by gradual dehydration. This holds true also for the cilia of the respiratory system (trachea) of the mammal.

4. The effect of cold in slowing, and finally paralyzing, the action of ciliated epithelium was abundantly verified for both amphibia and mammal.

5. Excess of moisture modifies the extent and direction of ciliary propulsion, by causing gravity currents which flow above those cilia produced, and in a direction determined by the shape and position of the part.

Consideration of the importance with which these principles may hold from the viewpoint of normal and abnormal physiology, especially of the respiratory apparatus, will be deferred until the second part of this research is completed.

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DISCUSSION.

Dr. R. A. Barlow, Madison, Wis.: A paper such as Dr. McDonald has just given us may be of inestimable value, depending on the degree of practical application which may be made of the same. It is this sort of research and investigation upon which we as clinicians must build up the rationale of our handling of cases in our offices. As Dr. McDonald has emphasized, the ciliated epithelium is a barrier to the invasion of the organism by the hordes of bacteria omnipresent on the respiratory mucosa. Any stimulation or retardation of ciliary action, either thermal, chemical or by any other agent, is bound to result in a manifestation of toxicity in the nature of the commonly called "cold" or, as we are now designating it, acute respiratory infection.

The usual treatment directed to the nose and throat in the average office, wherein markedly astringent drugs are employed, oftentimes may result more disastrously than beneficially. The immediate effect of such treatment, while apparently ameliorating the annoying symptoms of coryza, with its attendant stuffiness of the nose, is soon followed by a return of congestion of the tissue, as severe if not worse than before the treatment was instituted. Referring again to Dr. McDonald's observation, it seems inevitable that there will be a temporary retardation or paralysis of the ciliary action, sufficient at least to allow further ingress of bacterial products. Realizing that the socalled "cold" is a result of some form of indiscretion or deviation from our routine mode of living beyond reasonable limits, we can visualize disturbances of this ciliary motion. Rather than limit ourselves to local therapy, we should endeavor to reestablish the normal action of the respiratory mucosa by measures directed to the general well being of the individual.

Dr. McDonald's paper, as is true of all laboratory investigation, represents weeks and months of tireless, painstaking observation and research. He gives to us certain facts and data as the result of this work, trusting that we will accept it for what it is worth. The ultimate use of this material in our practical application of it will determine its clinical value.

I wish to congratulate Dr. McDonald, and to express the hope that our future programs will embody a greater number of papers of this type, as we are in need of a closer alliance between clinical medicine and laboratories of physiologic and biochemic research.

Dr. James F. McDonald, Omaha, Neb., (closing): I shall not give here an extended discussion as to the role of the principles we have established in reference to ciliary activity, in their application to physiologic and clinical problems. There are always many applications of fundamental principles. We are extending our research upon several aspects of this problem.

One of these is along the line of Dr. Barlow's suggestion, that the most safe and sufficient guide in the use of therapeutic agents, in the respiratory territory as elsewhere, is a knowledge of their physiologic and pharmacodynamic actions. In using therapeutic agents, we should know what they do to the human tissues as well as to invading the microorganisms, and how they do it. As yet, I believe, there is very little known about the effect upon the rate of protective ciliary propulsion of the many drugs used in the treatment of affections involving ciliated surfaces. One line of research in our laboratory is devoted to this prob-

lem, with the view of throwing light upon the important question of the effect of these chemical and physical agents upon protective ciliary functions. In the past, we have been too prone, it seems, to transfer the test tube action of physical or chemical substances, and assume that an efficient germicidal agent acting in vitro will give similar results safely and efficiently when applied to the tissues of the human body. In treating respiratory surfaces with strong disinfectants, we should be able to tell whether and to what extent serious physiologic or anatomic damage is also done to the dynamically protective propulsive ciliary mechanism. This subject is in urgent need of exhaustive research.

Another problem we are investigating is the effect of dryness of the air upon the incidence of respiratory infection. It is well known, that respiratory infections each year are responsible for much suffering and enormous loss, economically, as well as of health and even of life. It is further well known, that the amount of moisture in the air increases and diminishes with the rise and fall of temperature, being most humid during hot weather and least humid during cold.

For example, one cubic foot of air at $-20°$ C. can carry, when fully saturated, only 0.167 grain of water vapor; whereas at 100° F., a cubic foot of air will hold 19.966 grains of moisture, which is over 100 times the amount of water vapor present at the lower temperature, tho in each case the relative humidity is 100.

The air in our homes in cold weather is known to be very dry. On entering, the atmosphere contains, as we have seen, but little moisture. This is further reduced, relatively, on the inside by expansion due to heating without a sufficient addition of moisture, the humidifiers of the average heating plant being wholly inadequate. There is a still further moisture loss from inside air, by condensation upon the inner surfaces of the walls in cold weather. Of these causes, which all contribute to the drying of air, the factor of cold is most potent, because air coming from the outside in cold weather is always so dry that the further reduction tho important is relatively less in extent. Since the incidence of respiratory infection stands in such definite relationship to the fall of moisture content, and since drying paralyzes ciliary function, it is of the utmost importance for us to find out to what extent the moisture lack may play a direct etiologic role in the incidence of infection. Our research bears also upon this phase of the problem.

We are investigating also another point in relation to the principles set forth in this paper, that is, the effect of cold, per se, upon the incidence of respiratory infections. It has been known for a long time that cold slows the action of contractile tissue, including cilia in lower biologic forms. The senior author has shown, as noted in this paper, that cold has the same effect upon the cilia of the mammal. Cilia, therefore, are directly cooled and, it seems, correspondingly retarded in action by cold air when inhaled. There is a further cooling in the nose by the evaporation of moisture from nasal mucosa, and the drier the air when inhaled, the more moisture will it evaporate, and therefore the more heat will it abstract from the respiratory surfaces. The principle of the "absorption" of heat during the evaporation of a fluid is too well known to require discussion here. One other mechanism of nasal cooling should also be noted in this connection. It has been shown by the work of

Mudd, Grant, and others, that cooling the skin surface causes a cooling of the pharyngeal mucous membrane. The authors explain this cooling as depending upon a vasomotor reflex. The afferent nerves of the reflex arc here would be the temperature fibers of the skin, and the efferent, the autonomic vasoconstrictor nerves to the respiratory mucosa. It has been shown that the nasal lining and the external auditory meatus are similarly reflexly cooled from the cold applied to the skin.

It seems, then, that cold air, being dry, may paralyze cilia by dehydration, and further paralyze them by (1) direct cooling, (2) by cooling by evaporation, and (3) by cooling thru vasoconstrictor reflex from skin to the respiratory mucosa. The fact that cilia are paralyzed both by cold and drying, make it important that these two factors be exhaustively investigated in relation to the problem of respiratory infection. One important phase of this question which we are investigating is whether, and to what extent, the moisture and heat of the expired air may offset the paralytic effect of the dry, cold inspired air.

Something has been asked about the function of sinuses. In our work upon nasal cilia, we have had an opportunity to observe the action of cilia in the sinuses of living animals. The ciliary mechanism lining the sinuses is very active. One gets the impression that moisture secreted by the sinus is deposited in the nasal cavity thru the ostia. I have formulated a theory, as a guide to research, that these sinuses form an auxilliary irrigation system for the nose. Such a function would be in line with the physiologic principles noted in this paper. It would tend to furnish an adequate supply of moisture to prevent local dehydration paralysis of the cilia when, during inhalation of dry air, the amount of evaporation tends to exceed the moisture secreted by the glands of the nasal mucosa. It would also cooperate in the production of moisture during the "flushing" stage of colds, hay fever, etc. The older theories of function of the accessory nasal sinuses are not very satisfactory. We shall gather more data upon this point in the course of our research.

In view of the fact, that Nature uses a method of "flushing" in early respiratory infection, and, further, that the drying of cilia paralyzes them, the practice of treating "early colds" by what are called "rhinitis tablets," with the view of drying the respiratory mucosa may be open to serious criticism. Such treatment may involve the old fallacy, that blanketing a symptom which may be only the evidence of the activity of one of Nature's weapons against invading enemies, has per se a beneficial or curative value, whereas what we may actually be doing is forcing out of operation weapons which are vitally essential in the combat. It seems desirable that physiologists, pharmacologists, otorhinolaryngologists, etc., set into operation a coordinated system of research that will throw light upon problems like those discussed in this paper.

There are several implications from the principles set forth in this research that time does not here permit me to discuss.

It is an interesting situation, that, despite the fact that the respiratory portal permits in our climate a high percentage of the total incidence of infection to which we are subject, we yet know much less about the physiology, normal and abnormal, and the pharmacodynamics of the protective respiratory mechanism than most of the other body systems.

STENOSIS OF THE LARYNX.

R. C. Lynch, M.D.

NEW ORLEANS, LOUISIANA.

In opening this discussion on stenosis of the larynx, I would like to have your permission to begin at the wrong end first. (Bibliography.)

I do not care to discuss any form of acute laryngotracheal stenosis or the congenital stenosis seen in the new born or held over until later in life—webs, malformations and deformities. Also neoplastic or paralytic stenosis is not under consideration. In other words, my effort is to limit this discussion from an etiologic standpoint to scar tissue stenosis—if I may use that term—for in my experience—exclusive of tumors and tuberculosis at least—90% of my cases fall under that head.

From an etiologic standpoint, it would seem that all scar looks alike, and yet the traumatic scar and results therefrom and the scar from disease produce decidedly different results, so that these two classes will offer you an opportunity to discuss your cases more systematically.

I have always looked upon the stenotic larynx as a funnel, with the small end usually at the subglottic space or just below the vocal chords. Now in the traumatic cases, it is usually the upper end of the funnel that is most damaged, namely, thyroid cartilage-arytenoid joints, even epiglottis, so that in these cases, in addition to the usual narrowing of the lumen from granulation, web hypertrophy or hyperplasia, one has to combat a very formidable situation in displacement of the thyroid and arytenoid cartilages, while in the stenosis from disease, one usually has to deal with a lesion involving the anterior half of the cricoid cartilage as a result of a hurry up tracheotomy, the outcome of which is cutting the cricoid cartilage and upper rings of the trachea, not only thru, but out of the middle line. These cases of either class usually present themselves with a tracheotomy or sadly in need of one.

The first essential is to be sure that the tracheotomy is placed low and in the middle line; after that has been done, the patient

accustomed to the tube in its new location and the wound healed, we are ready to tackle the problem of the stenosis.

Since the patient comes with the diagnosis made and we know the etiology and pathology of scar tissue, the treatment becomes the most important factor in the case after all.

What are the essentials for treatment, may I tabulate for brevity?

1. Sympathy of hospital organization from superintendent to orderly.
2. Patience, perserverance, courage of conviction and plenty of time on part of operator.
3. Hospitalization of patient.
4. Thoro diagnosis as basis for plane of attack.
5. A good tool box. (Lantern slide to show all that I use in this work.)
6. Webs are atrophic and hypertrophic: the former are dissected thru with knife and forceps or cut with knife or double curet; the latter must have some means to keep the raw surfaces apart—a narrow intubation tube or preferably a piece of hard rubber, wired in place by putting a wire thru the thyrohyoid membrane, another thru the cricothyroid membrane and tightened over a lead plate. By this means pressure is made anteriorly, and if left in place two weeks, healing should be complete.

Hypertrophic areas, whether arytenoid, cordal or subglottic, yield best to a fairly deep cautery cut. I use Yankauer's tips. At least 85 per cent of the cases come with a history of diphtheria, increasing laryngeal stenosis, intubation, which gives relief, then the tube is coughed out and must be replaced. This occurs two to five or six times, until replacement of intubation tube is impossible and quick tracheotomy is done—always too high, and rarely if ever straight. Decanulation after this storm is impossible, and the case presents itself with funnel larynx, arytenoids more or less fixed, cords and subglottic space in one mass of scar, with usually a tiny hole size of a pin at the most posterior portion of the scar. Having replaced the tracheotomy and given time for healing, the interval is spent in building up resistance and acquiring hospital habits. All intralaryngeal manipulations are done under suspension, and all operative works of placing tubes and so on under general anesthesia.

Dilatation is accomplished beginning with filiform rubber tubes, metal tubes of block tin of special length, diameter and

shape to suit the case, covered or not with rubber tubes, and finally intubation tubes covered or not with rubber. The action of rubber on scar tissue is well known; hot bougies as is used in the esophagus has not given the same degree of softening in the larynx.

The next problem is straightening the lumen of the laryngotracheal pathway. This refers to spurs developed posteriorly, collapse of the anterior wall, and is always necessary in the traumatic cases, such as automobile accidents, fractures, and suicidal cuts. Here the deep ignipuncture of the French is extremely useful in taking down overhanging redundant hypertrophic tissue, and I have done plastic operations with flaps, cuts and sutures with very nice results. The pathway is straightened finally by the long intubation tube, using usually the hard rubber tube, to be left in place from three to six weeks and sometimes to be worn from three to six months and sometimes longer.

Finally, the greatest help in the worst cases comes from thyrotracheotomy, with the area laid wide open for inspection.

In house cleaning, so to speak, the furniture can be by submucous resection, dissection, replacement and alignment, put back as near to its original place as possible. Rubber tubes, vaselin worsted or gauze hold the parts in the new position, to be replaced later by hard rubber tube posts, etc., to bring about the desired result.

If laryngostomy with permanent fistulae or opening is aimed at, the technic is so admirably described by Jackson in his peroral endoscopy and laryngeal surgery that I need not make further mention, except to say that in my hands it is hard to accomplish, and only twice have I succeeded. The remainder, however, were brought to final mouth breathing by combined thyrotracheotomy and intubation.

Dr. Arbuckle has grafted one or two cases, and as he is to open the discussion I will only mention it.

In conclusion, these cases try the patience of Job, and in many cases, many weeks' work are lost because of an intercurrent grippe, pneumonia or intestinal flare up.

The patient must acquire a state of immunity to the reaction of inflammation. In some, this seems developed; in others, with each new laryngeal manipulation will come a new generalized inflammation, which not only spoils the local progress, but will jeopardize the life of the patient. The final prognosis is good, provided one can keep his patient alive. This sounds like a foolish statement, but it is true.

Then, too, one must expect hairbreadth escapes on the part of the patient from a slipping tube, either tracheotomy or intubation, hemorrhage or shock. It is very essential that nurses and staff men be acquainted with the quickest way to reestablish breathway, and with each manipulation, I always tell interne and nurse how best to proceed if necessary. This has saved many a case in my experience.

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DISCUSSION.

Dr. Millard F. Arbuckle, St. Louis, Mo.: Dr. Lynch has well said that these conditions are among the most difficult with which we as otolaryngologists are confronted. In the first place, they are very serious, because they interfere with the essential function of respiration. As Dr. Lynch has pointed out, if anything material goes wrong, the patient will likely lose his life, unless there is someone at hand who understands what to do and does it immediately.

My experience has been the same as Dr. Lynch's, in that the great majority of these cases are caused by diphtheria. However, I believe more of them than we realize are caused by trauma, or by a cut throat from suicidal or homicidal intent, gun shots, accidents, falls, etc.

The treatment is a very difficult problem. The type to be used must depend to a great extent upon the type of lesion present, which, as you know, may be a combined lesion. We may have the supraglottic hypertrophic type, or the infraglottic hypertrophic type, as well as webs and spurs and dislocations. In addition to these organic types, we may have the socalled neurotic type, the patient who is afraid to have the tube removed, and while his airway is perfectly good so far as mechanical obstruction is concerned, he may have an attack of fright and a spasm purely from fright, which will kill him just as dead as a web across the larynx. So we must be guided in our treatment by the type of lesion present. The hypertrophic type is best dealt with by the electrocautery and by pressure, as is fully described in Dr. Jackson's work. The neurotic type requires a good deal of maneuvering as well as medical skill. Then, we have a type in which there has been a perichondritis with sloughing of a considerable part of the thyroid cartilage and occasionally the cricoid cartilage, in which there is deformity and displacement of the wall of the

larynx, with adhesions. Some of these cases have complete obstruction. In my experience, I have been unable to get anywhere in these cases with the ordinary endoscopic methods.

One peculiar thing I have noticed in the cases which I have seen, is the fact that all the cases had destruction of the cartilage on the left side. When I noted that, I took the trouble to find out whether or not they had been intubated by a righthanded man, and they had. I do not know what caused the perichondritis, but it came after trauma and diphtheria.

I have had three cases of which I will show slides. Instead of trying to dilate them after I found they had complete obstruction, I dissected away the scar tissue, replacing the walls as well as I could. A tracheotomy, of course, had already been done. The denuded area was covered by Thiersch grafts, held in position by sea sponge cut to proper size to fill the lumen snugly, and anchored by thru-and-thru suture. The wound is closed and left for eight days.

These cases have been done for three and four years. One child died, and the other two were perfectly successful. There was no treatment required after the skin graft was put in position, and the lumen seems to be perfectly good. It can now be seen with a mirror.

Dr. Gabriel Tucker, Philadelphia, Pa.: A case came into the Clinic recently, from which we learned a lesson that might be of interest. This little fellow, under one year of age, came in with a complete closure of the larynx, postdiphtheritic. We tried to put in a tracheotomy tube, and finally succeeded, by using the Jackson direct laryngoscope, in getting thru the larynx. It was several weeks before we could get a string thru and draw it out at the tracheotomy wound. Here it is alongside the tube, carrying the end on thru and out his nose. Then we pulled a very small sized tube back thru the larynx and finally got a small opening. Following this, we used Dr. Jackson's method of dilating the larynx by increasing the size of the bougie.

The point I want to make about this case, is the age of the child. At this age, it is almost impossible to run anything thru the trachea or larynx. It will increase the stenosis. Laryngectomy is out of the question. We feel, that if this method is carried out in recent cases of stenosis, cases that develop soon, it will prevent a great many very serious cases of postdiphtheritic laryngeal stenosis.

Going back to this matter of dilatation, as you dilate the larynx, the child is accustomed to breathe thru the larynx; then we place a cork in the tracheotomy tube, so he gets part of the air thru the tube and part thru the larynx, and in that way he is reeducated. Finally, after he can breathe easily, we use a cork of large diameter, and after a while a full sized cork. After a while, the patient will be able to wear a complete cork day and night. We use the cork for six months.

As to laryngoscopy with other methods, we split the larynx, excise the scar tissue, and put in first an iodoform pack and a tracheotomy tube below it. I will show you the technic of this special apparatus. It is fool proof. When the tracheotomy tube is removed, the patient removes the dressing from the neck and takes out the plug. There is no possibility of anything going wrong if the instructions are followed. I will show you how it is put together. First, we use a gauze pack until the larynx is completely epithelialized, and we cover this pack with a finger

cot—a glove finger. After three or four months, we make a pack with a pinehole drain in the center, and cover it with a finger cot as you see it here. In order to maintain this plug in position, we sew a strip of gauze on the side of the finger cot. We put this plug out thru the larynx, letting the two ends of the gauze come across the neck. Here you have the long ends of gauze and the plug in the larynx above the tracheotomy tube. There is tied to the end of the string a small pad of gauze, then adhesive tape is put on either side of the neck. If the dressing is taken away, it brings the plug with it, because the gauze is sewed securely to the plug.

We find this dressing is less irritating than any other type, pure rubber being less irritating than a piece of rubber tubing.

Dr. Robert C. Lynch, New Orleans, La., (closing): I was in hopes that more of you would take advantage of the opportunity to discuss these terrible cases that we have to deal with, because each one of you must have some device which would help us all. I wrote the paper with that idea in view.

Dr. Arbuckle's idea of grafting the larynx and trachea is certainly to be commented upon favorably, and I think his results speak for themselves. He is to be congratulated on his results.

Dr. Tucker brought out one point which I should like to emphasize, and that is, that if you cannot treat these cases yourself, send them to somebody who can, and do it early. The earlier we can get hold of cases of this kind after the fact of decannulation is established, the more likely are we to save the patient's life. The method of decannulation with a cork offered by Dr. Tucker has been known—Dr. Jackson gave it to us a long time ago, and it represents the method par excellence for the final determination whether the lumen of the larynx is sufficient to keep up respiration. Certainly, it is advisable to keep the cork in at least six months before decannulation is attempted.

The method of treatment by laryngostomy, as I said before, in my hands is difficult to accomplish. I spent some time with Dr. Patterson, ten or fifteen years ago, watching her dressing these cases under Dr. Jackson's supervision, and learned to carry out the dressing as it was then practiced. I see some improvements which they have made since then, and which Dr. Tucker has brought us today, and I found, at least for myself, a very valuable suggestion in the slides he has thrown on the screen.

THE ADVANTAGES OF THE MODIFIED LOTHROP OPERATION OVER THE KILLIAN PROCEDURE IN THE TREATMENT OF CHRONIC FRONTAL SINUSITIS.*

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In compiling this thesis, I have not relied on personal observations solely, but have drawn from the experiences of the other members of our Clinic as well, so in citing cases, I shall use the material of the entire clinic.

First let us go over the different operations, namely: the Killian, modified Killian, the Lothrop and its modification, as done in the Skillern Clinic.

The Killian, as first described and performed by Killian,[2] had as its object the obliteration of the frontal sinus by allowing the periorbital tissue to ascend from below, and to apply the skin and subcutaneous tissue, originally in front, to the posterior wall, thus avoiding or rather minimizing a distinct disfiguration, and at the same time permitting an exenteration of the ethmoid and opening the sphenoid cells without danger of penetrating the cranial cavity.

TECHNIC: A preliminary intranasal operation to remove all polyps and hypertrophies dependent upon the sinus infection must be done, and skiagraphic films made to determine the size and shape of the sinuses. (At this point I wish to bring out the importance of obtaining clear, sharp negatives, that may be studied in the stereoscope. A stereoscope examination will not only show the depth of the sinuses, but will often bring to view small, infected, misplaced ethmoidal cells. If these infected cells are overlooked, they will be the cause of the failure, which otherwise would have been an excellent result.) Under a general anesthesia (Killian using chloroform), the nasal cavity of the affected side is plugged with four cotton tampons, the size of a cigar, attached to silk sutures. These tampons are packed, one

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

in the olfactory fissure, a second in the middle nasal fossa, a third on the floor of the nasal cavity, and the last between the ascending process of the maxillary bone and the septum. This last tampon was supposed to support the mucosa and catch the pus, also prevent injury during the resection of the bone.

After cleansing, an incision is made thru the center of the eyebrow, beginning at the outer end. When the nasal end is reached, the incision is carried downward in a curve alongside the nose to end at the base of the nasal bone, the eye being protected by a gauze pad. For accurate approximation of the wound, several small cross incisions are made as markers.

A rather profuse hemorrhage occurs at this point, which is arrested by hemostats. Edges of the wound are undermined and retracted, so that the periosteal incision may be made 6-8 mm. above and parallel to the supraorbital ridge. The second periosteal incision is made, slightly internal to the supraorbital notch and extending downward thru the center of the ascending process of the superior maxillary. The periosteum above the supraorbital ridge, being elevated, is retracted, and a groove is made in the bone with a V shaped (Killian) chisel, following the line of the periosteal incision, until the sinus is penetrated. The lower surface of the groove forms the upper edge of the supraorbital bridge of the bone. All of the anterior wall of the sinus lying above this bridge is removed by hammer and chisel or bone forceps. The mucous membrane and all partial septa are thoroly curetted, special care being given to all recesses and hollows. The bridge is smoothed off and the sinus loosely packed with gauze.

The resection for the lower opening is now undertaken: the periosteum is elevated from the frontal process, lacrimal fossae and orbital portion of the frontal bone, almost up to the supraorbital notch.

A groove is cut thru the suture formed by the nasal bone and frontal process of the superior maxillary in an upward direction by means of the Killian chisel; another groove is made thru the frontal process below at right angles to the first, care being taken not to injure the lacrimal sac or underlying nasal mucosa; a third groove is advisable above, extending outward thru the nasofrontal suture, forming the lower edge of the bridge. Neglecting to make this third groove may prove disastrous: when prying out the resected portion of the ascending maxillary process, you may find that you have also broken your supra-

orbital bridge, especially if there be a deep supraorbital notch. A small opening with a half round chisel is made at the junction of the grooves, and the bone removed piecemeal, so as not to lacerate the nasal mucosa, which is to form the flap leading into the frontal sinus.

The limits for the resection are: above, trochlear attachment, supraorbital notch; behind, the anterior ethmoidal vessels; below, lower part of the lacrimal groove.

After the orbital tissues are retracted, the anterior and posterior ethmoidal cells may be exenterated to the anterior wall of the sphenoid. This also may be removed if the sphenoid is diseased; reflected light will be necessary for this procedure.

The resection of the nasal mucosa beneath the resected frontal process and turning it into the sinus, completes the operation. The wound is flushed out with normal salt solution, iodoform insufflated, and the edges are approximated with aluminum-bronze sutures. A gauze strip in the nose which extends upward holds the flap of the mucosa in position. This strip is removed on the second day and the sutures on the fifth.

Killian used moist boric acid gauze dressing. The main drawbacks of the original Killian operation were: First, the time required—It took from 1½ to 3½ hours. Second, the difficulty in technic—i. e., resection of the maxillary process without injury to the nasal mucosa or the trochlear yet reaching the outermost recesses of the sinus.

These drawbacks to the original operation were soon found to be unnecessary, as experience taught that the superior oblique muscle and trochlear[2] nerve could be loosened from their positions and retracted without fear of permanent diplopia, provided they were carefully brought back to their original position[3] and a stay suture of catgut so placed as to hold them in situ until healing occurred. It was also found that the careful resection of the ascending maxillary process to conserve the mucous flap was entirely unnecessary, as the case did just as well when the flap was either destroyed or lacking as when carefully packed in place. With these two procedures—so emphasized by Killian—omitted, the time of the operation was materially shortened and the operation of choice became the socalled modified Killian, with slight variations in the technic, according to the opinions and results of the different operators.

I shall not attempt to enumerate or describe the many different modifications of the Killian operation, but will confine my

remarks to the modified Killian as prescribed and taught in the Graduate School at Pennsylvania.

The stereoscopic pictures studied and all abnormalities marked, the preliminary intranasal operation for the removal of the anterior end of the middle turbinate, hypertrophies and polyps having been done at a previous sitting, and the patient prepared for a general anesthesia, he is sent to the operating room where morphin sulph., gr. 1/8, atropin, gr. 1/150, is administered by hypodermic. Induction by gas-oxygen is followed by ether. As soon as the patient is quiet, the head is securely bound with a sterile towel, just below the hair line. In a female, it is important to see that no wisps of hair escape.

The eye being protected, the entire field is given a heavy coating of iodin. This includes from the upper eyelid and side of nose to the hairline, especial attention being given to the eyebrow. A practice is made in this clinic of clipping both eye brows with curved scissors, for the following reason: sterility of the part, and that both eyebrows will grow in the same length and color. The other side is prepared in case incision thru it is found necessary. Shaving, especially of one eyebrow, has been entirely done away with, as after shaving the eyebrow, one is never certain how it will grow in, whether light or so heavy and long, as to be out of all proportion to its fellow.

The forehead and operative field is now cleansed of the iodin with an alcohol sponge, and sterile linen draped, and head braced by sand bags. Two c.c. of adrenalin chlorid, 1:1000, is injected hypodermically along the line of incision thru the eyebrow and down the side of the nose. The majority of the solution is injected around the lacrimal fossa, beneath the periosteum, as it is here our most troublesome hemorrhage occurs. No intranasal packing is used, any blood that may later escape into the choana being removed by suction.

After a wait of several minutes (3 to 5) for the adrenalin to take effect, the incision is made. For the right side, the incision should be started at the eyebrow and carried downward; for the left, from the cheek upward to the eyebrow: it is then carried outward thru the eyebrow to within an eighth of an inch of the end of the hairline. (Several small cross incisions are made as markers for closure.) The incision should be carried down to, but not thru, the periosteum. The hemorrhage is controlled by hemostats, a pad being placed over the eye to protect it from undue pressure from the hemostats.

The soft parts are dissected away from the periosteum, above the orbital ridge, and both above and below the attachment of the lacrimal bone with the ascending process of the superior maxillary; the periosteum is incised one-half inch above the supraorbital ridge, and the upper edge elevated for a space of two centimeters. The lower edge is merely raised to prevent its injury by the chisel when the groove is made. A small round opening is made with the half round chisel in the anterior wall of the sinus above the supraorbital ridge, immediately above the frontal articulation of the superior maxillary. A study of the X-ray plate for position of the septum will show the central limitations of the sinus.

After the bone has been penetrated, a bent silver probe is introduced and carried in all directions so that the various dimensions of the sinus may be ascertained.

The periosteum is elevated upward, slightly beyond the external confines of the sinus, and inward and downward the length of the original external incision, but sufficiently internal to allow the formation of a bony bridge. Care must be exercised not to loosen or disturb the periosteum to within a half inch of the supraorbital ridge, otherwise it is elevated slightly beyond the boundaries of the sinus in all directions.

The periosteum and soft parts are now retracted by an assistant, and with the angular V chisel, a furrow is made along the line of the periosteal incision, which will form the superior edge of the bridge. It is just as well to make this furrow a little higher up than the incision above the supraorbital ridge, as the wider one makes the bridge, the less likelihood there is of breaking it during later instrumentation. Any excess can easily be trimmed away with bone cutting forceps, when making the final inspection before closure. Another advantage in making the furrow in the bone higher than the periosteal incision, is the ability to bring the periosteal flap down over the upper edge of the bridge, thus insuring it a better blood supply and minimizing the later disfigurement by the sinking of the skin above the bridge.

The furrow into the bony wall is made by placing the chisel at the outer limit (as found by probing) of the sinus, three-quarters of an inch above the orbital rim, and by careful tapping with the hammer, keeping a like distance from the orbital rim, cutting a shallow curved furrow ending at the round opening made above the frontal articulation of the superior maxillary. A flat chisel is now used to enlarge the furrow to a slit in the

sinus, by gently clipping away the upper edge. The entire anterior wall is removed piecemeal with larger hollow chisels or suitable rongeurs, until the sinus is completely bared. Particular attention is paid to open all ramifications in their fullest extent, and all partial septa are removed.

The diseased portion of the sinus mucosa is next removed with a curette, and the underlying bone minutely inspected for any traces of necrosis. The X-ray plates are referred to at this point to recall any abnormalities, such as septal cells, which, if present, are opened and curetted.

Hemorrhage is controlled by plain gauze pack, while the inferior wall is attacked as follows: the periosteum is incised at the upper internal angular edge of the orbital ridge, the incision being carried down to the lower part of the attachment of the lacrimal bone with the ascending process of the superior maxillary. The edge of the periosteum is carefully elevated, then, together with the attachment of the superior oblique and trochlear nerve, is raised in mass and pushed over the orbital fat.

The lacrimal duct is raised from its fossa along with the periosteum lying over the anterior third of the lamina papyracea. As much of the internal and superior orbital socket is laid bare as found necessary to give working space. A troublesome hemorrhage follows, due to injury to the ethmoidal vessels, which is controlled by packing with plain gauze wrung out in 1:1000 adrenalin chlorid and allowed to remain for several minutes.

Hemorrhage being controlled and the wound cleaned, the sharp angle chisel is placed at the superior internal angle of the orbit beneath the sinus floor. A light tap of the hammer is usually sufficient to make the opening into the sinus, which is enlarged to permit introduction of bone forceps for biting out the floor. It is best to use a straight chisel when nearing the osseous bridge, in order to avoid breaking into it. The entire floor of the sinus is thus removed.

From this point of the operation until the end, the patient's breathing must be noted for signs of blood in the throat, and the suction used from time to time.

It is next necessary to remove a portion of the ascending maxillary process, in order to reach the infundibular cells and have access to the bulla, middle turbinate, etc., so the periosteum is elevated and two horizontal furrows are made in the ascending process opposite and anterior to the lacrimal bone. A large concave chisel is used to lift out the bone between the furrows, thus

laying bare the anterior ethmoidal structures. The superior portion of the lacrimal bone is removed by bone forceps, the flap of underlying nasal mucosa trimmed, the bulla and entire anterior ethmoidal labyrinth exenterated back to the lamella of the middle turbinate.

In a case in which the posterior ethmoid and sphenoid sinuses are involved, it is a simple matter to continue the extirpation of the ethmoidal cells and remove the anterior wall of the sphenoid.

A strip of gauze packing is introduced into the nose in the direction of the frontal sinus, to determine whether the communication is sufficiently extensive. If not, the anterior superior nasal spine is removed with the bayonet chisel. The gauze packing is sawed back and forth a few times, to remove rough edges and to enmesh in its folds any loose spicules of bone. The upper edge of the bony wall of the sinus is now bevelled, to prevent a sharp sinking in of the tissue at this point. It also gives a little more periosteum to bring down when suturing.

Toilet of the wound is made by moist sponge or irrigation with normal saline solution, and final inspection for bony spicules and debris.

A piece of rubber tubing somewhat smaller than a cigarette is now inserted into the nose and brought out of the sinus, the end trimmed at an angle and placed about the center of what was the floor of the sinus, and the wound is ready for closure.

The superior oblique is sought, and a No. 2 plain catgut suture, in a curved round needle, passed under it, taking a good bite into the periorbital fat. This is brought up and tied to the inner angle of the bony bridge. It may be necessary to make a shallow notch in the upper edge of the bridge at this point, to hold the suture in position. This procedure will prevent diplopia, which is very apt to occur, if omitted.

If the sinus has been a large one and extended well out over the eye, a small wick of gauze is placed in the outer angle and brought out at the outermost edge of the wound.

The upper flap of periosteum is brought down and sutured to the bridge with No. 1 plain catgut. This prevents, to a great extent, the sinking in of the anterior wall at an acute angle, and hence lessens the deformity.

Some may say this prevents one of Killian's main objects, i. e., obliterating the sinus by allowing the skin and subcutaneous tissue, originally in front, to adhere to the posterior wall of the

sinus. That is true, but just as good results are obtained with less deformity, and it is results we are after.

The subcutaneous tissue and skin is closed with silkworm gut, care being taken to bring down a sufficient amount of subcutaneous tissue to cover the wound entirely, and that the marker incisions are brought in opposition. Three layers of a strip of iodoform gauze are placed over the incision, gauze pads placed over the eye, and fluffed gauze over the wound, which is held by gauze bandage.

The gauze pads covering the eye are moistened every two hours with the following solution:

℞ Liquoris plumbi subacetatis dil. f℥ ii
Tincturae opii
Tincturae belladonnae $\overline{aa}$ f℥ ss
Tincturae arnicae
Aquae destillatae aa q.s. ad. f℥ iv

This seems to be a great comfort to the patient, lessening the pain and inflammation in the orbit.

The after treatment consists of having patient lie on the sound side, and refrain from blowing the nose. The following day, dressings are removed and all blood drainage wiped away with bichlorid solution, 1:5000, and fresh dressing applied.

The wound is not dressed again until the third day, unless the patient complains of discomfort from the dried gauze, or untoward symptoms, such as fever above 101°, or severe headache develop. The wound is then dressed daily, and stitches removed after fourth day (if wound has healed). The rubber drainage tube is left in place for 10 days to allow for cicatrization to occur around it; it is then gradually removed, cutting off ¼ inch daily until it falls from the nose of its own weight.

By allowing cicatrization around the tube instead of across the wound, as would occur if the drainage tube were removed earlier, there is formed an ostium of scar tissue which remains patulent.

Lothrop's Operation.

From a rhinologic view, this operation has several disadvantages: i. e., the opening of a healthy sinus, and the unnecessary sacrifice of a portion of the septal nasal wall.

As the original Lothrop operation is not done in our clinic,

I shall merely touch on the salient points, giving in greater detail the steps in the modification of this method.

A preliminary intranasal removal of the anterior end of the middle turbinate and anterior ethmoidal cells is necessary.

An incision similar to the Killian, but not as extensive, is made, beginning at the center of the eyebrow and curving downward for an inch beyond the eyebrow, along the side of the nose.

The soft parts and periosteum are elevated above and below the incision, and the sinus entered just above the suture of nasal process of the maxillary and frontal bones.

The sinus is gently curetted of all inflammatory tissue, the periosteum being left in situ. The cavity is explored with a probe to determine the location of the ostium. This is sounded and the probe brought downward into the nose and out of the anterior nares.

The ostium is enlarged by curettes and drills from below forward. Care must be exercised not to injure the cribiform plate. A small electrically driven burr is used intranasally for removal of the anterior nasal process. Always cutting toward the nasal bone and nasal process, at the suture line with the frontal, as the opening increases in size, larger burrs may be used, until the entire anterior nasal process is removed. The remaining ethmoid cells around the infundibulum, lacrimal bone and agger nasi are broken down and removed.

A large portion of the interfrontal septum is removed, along with that portion of the nasal septum just below the sinuses for an inch or more.

The external wound is closed, without drainage, and compress bandage applied. There is the usual treatment of wound, irrigations being avoided for several days.

The Modified Lothrop.

The same preparatory technic is used for this operation as that for the modified Killian. Skiagraphic plates having been made and studied in the stereoscope, and all polyps and hypertrophies having been removed at a previous sitting, we are ready to proceed with the major operation.

For the right side: The incision is started at a point about one half inch below the inner end of the eyebrow, over the suture line of the nasal bone and ascending process of the superior maxillary. The incision is carried upward and out-

ward, in a graceful curved line, thru the center of the eyebrow to its outer extremity (Fig. 1).

For the left side: The incision is started at the outer end of the eyebrow and brought inward, care being used to remain in the center, or just below the center, of the hair line. When

Plate No. 1.
Fig. 1. 1st Step. Skin incision for modified Lothrop operation.

the inner end of the eyebrow is reached, the incision is curved downward to extend down the side of the nose for about one half inch. This original incision is made very superficial, being more of a scratch on the skin, to give the line of future scar. Several small cross incisions are made, the chief marker being just below the inner end of the eyebrow. It is essential to make these markers very small and superficial if one wishes to minimize the scar.

Plate No. 2.
Fig. 2. Periosteum, incision above supraorbital rim and point of entering the sinus.

The incision is now carried down thru the skin, which is dissected free from the underlying tissue as high as the upper end of the sinus, and well over the mid line of the forehead. The incision is next carried down to, but not thru, the periosteum, and the muscle and fascia dissected upward and inward (Fig. 2).

A troublesome hemorrhage from the angular branch of the lateralis nasi and from the frontal and supraorbital will require ligation with No. 0 plain catgut. Other bleeding is controlled with hemostats and gauze packing, wrung out in adrenalin, care being used to protect the eye from the pressure of the hemostats.

The skin and underlying tissue is next retracted, with four prong retractors, upward and inward, so that a point one half inch above the supraorbital ridge is exposed. While the retractors are held by the second assistant, the periosteum is incised.

Starting a quarter of an inch above, and remaining a like distance, the incision is carried outward the length of the supraorbital ridge. The periosteum is elevated upward and inward for about a fourth of an inch; the lower portion, or that over the supraorbital ridge, is not disturbed excepting along the very edge of the incision, which is elevated to prevent injury when the chisel is being used.

Plate No. 3.

Plate No. 4.

Fig. 3. Sounding for limitations of the sinus.
Fig. 4. Sounding outer limitations marking for bone incision.

With a small half round chisel, an opening is made above the supraorbital ridge immediately above the frontal articulation of the superior maxillary. By cutting directly downward, a button of bone the diameter of the chisel is outlined and pried out, leaving a small round opening for exploration of the sinus (Figs. 3 and 4).

The sinus is explored in all directions by means of a blunt soft silver probe, and its limitations noted. The periosteum is then elevated upward and inward slightly beyond the confines of the sinus. The next step would be the making of the bone incision, but as a precautionary measure against starting the opening beyond the outer limitation of the sinus, into the cranial cavity, the probe is again passed outward and the outer confine of the sinus marked above the supraorbital ridge, where the groove is to be started.

The angular V chisel is used to make the groove: this is done by placing the point of the chisel at the mark showing the external limit of the sinus and one half inch above the supraorbital ridge (Fig. 5).

Plate No. 5.

Fig. 5. Periosteum elevated upward. Groove being made in the bone, forming the superior edge of bridge. Note periosteum on the bridge is not disturbed.

By gently tapping with a hammer, a shallow furrow is cut in the bone, ending at the round opening previously made in the sinus for exploration, care being used to follow and keep a like distance above the orbital rim. The furrow is gradually enlarged until a long slit is made into the sinus.

A straight, broad chisel is next used, but only on the upper edge of the slit, to enlarge the opening, so that rongeurs or other suitable bone biting instruments can be used.

The entire anterior wall of the sinus is thus removed, from

its outer and upper confine to the intersinus septum, being careful not to injure the supraorbital ridge.

The diseased portion of the sinus mucosa is removed with curettes, and the underlying bone minutely inspected for any signs of necrosis. All ramifications are opened, and all partial septa removed, thus making the sinus one large smooth cavity (Fig. 6).

Plate No. 6.
Fig. 6. The anterior wall of sinus removed

The X-ray plates are referred to and studied in conjunction with the opened sinus. Any abnormalities shown by the plates are explored and, if found, opened in their entirety, thus throwing them into the sinus cavity.

Possibly the cell found most frequently is a supraorbital ethmoidal cell. It may be of any size from a pea to a long slit like cell, extending outward the full length, or even beyond, the floor of the frontal sinus. If this cell is situated in the outer half, it may have a long, tortuous, constricted, canal like ostium emptying into the superior portion of the middle nasal passage directly behind the frontal ostium.

Possibly the next in frequency are the infundibular cells, frontal bulla, cells in the agger nasi and septal cells, the last being more in the class of ramifications of the frontal. If any of these cells be diseased, as they so often are in a frontal sinusitis, and are overlooked at the time of operation, our procedure for a radical cure will in all probability be a failure.

The next step is the enlargement of the frontal ostium. A reflected light, or head light, is useful at this point for the

locating and sounding of the ostium. It may be necessary to enlarge the opening into the frontal sinus by the removal of more of the anterior plate downward and inward, to the naso-frontal suture. As the supraorbital notch is in this field and may be rather deep, it is well to leave as much bone as possible near the orbital rim, so as to avoid fracturing the supraorbital ridge (Figs. 7 and 8).

Plate No. 7.

Plate No. 8.

Fig. 7. Enlargement of ostium by rasp.

Fig. 8. Enlargement of ostium by rasp forward and outward.

The sound is passed thru the ostium and brought out the anterior nares. A rasp is guided down alongside of the sound and the sound removed. The ostium is now enlarged forward toward the septum, and forward and outward. The point of the half round bayonet chisel is placed against the anterior superior nasal process, and with gentle blows, this is broken thru and removed. All rough edges are rasped, and a long piece of gauze carried thru and brought out of the anterior nares. With a sawing motion, this gauze is pulled thru several times to collect all fragments of bone and loose mucous membrane in its meshes (Figs. 9 and 10).

This newly formed ostium should be large enough to admit a piece of rubber tubing the size of a lead pencil.

A procedure that I think originated in the Skillern Clinic is next carried out: The bevelling of all edges of bone around the external opening. This is done for the reasons as enumerated under the modified Killian operation, i. e., to do away with

a sharp edge with a sudden sinking in of the tissues, thus minimizing the disfigurement, and to give more periosteum. The entire sinus is wiped out with gauze, especial attention being given to all recesses, and, with a final inspection, the wound is ready for closure.

Plate No. 9.

Plate No. 10.

Fig. 9. Removing the anterior superior nasal spine from above downward.
Fig. 10. Bone work completed showing enlarged ostium. Note floor of sinus removed.

A piece of rubber drainage is carried down thru the opening into the nose and brought out the nares. The upper end, being bevelled, is placed about the center of the floor of the sinus and anchored by No. 2 chromic gut suture, the ends of which are brought out thru the skin and held by a hemostat.

Plate No. 11.

The periosteum is brought down by interrupted No. 1 catgut sutures, and the skin and superficial fascia with silkworm gut. The stay suture of the drainage tube is then tied over a hemostat and the ends tied long to differentiate it. The tube is then pulled down until the stay suture is tight; this should bring the upper end of the tube in the most favorable position for drainage. If there is a deep, gutter like supraorbital cell, which has been opened into the frontal sinus, the drainage tube is wedged down in the gutter, which will hold it in place and the stay suture will not be necessary. The other end of the drainage tube is cut off even with the nares. Several layers of iodoform gauze are laid over the wound, which is then dressed with sterile pads and bandage. The dressings are changed the following day and the same after treatment carried out as described for the modified Killian (Figs. 12 and 13).

Plate No. 12.

Plate No. 13.

Fig. 12. Periosteum, incision above supraorbital rim and point of entering the sinus. Closed by interrupted No. 0 catgut stutures.
Fig. 13. Stay Suture brought thru and tied. wound closed, catgut for periosteum, silkworm for skin.

In those cases of suspicious double frontal sinusitis in which a definite diagnosis has been made for the one side, but in which the condition of the other sinus is doubtful, it is good policy to break thru the thin intersinus septum with a small round chisel and enlarge the opening downward and forward until sufficient bone is removed to allow thoro inspection of the mucosa by means of a reflected light. If it is found to be diseased, it is quite possible, in some cases, to curette this sinus without further external surgery. Here let me emphasize,

that unless *all* diseased tissue is found and removed, one need not expect a complete and permanent cure.

If the sinus is found to be in a healthy condition, no harm has been done, as we have never yet had the infection extend from its diseased fellow.

In cases of double frontal sinusitis in which the adjacent sinus cannot be curetted thru the breach in the septal wall, it is a simple matter to procure complete exposure of the entire anterior plates by making an incision across the bridge of the nose joining the two lateral nasal incisions, and dissecting the entire flap upward. As the great majority of patients requiring a double frontal operation wear glasses, the cross incision is made where the nose piece of the glasses rests. If this is done, with a careful original incision and closure of the wound, the scar will be so insignificant that it will require very close observation to discover it, and will never be seen when the patient wears his glasses. If it is impossible to get sufficient space in operating on a very large single sinus, no hesitancy need be felt in following this procedure.

Let us next consider the relative dangers of the Modified Lothrop to the Modified Killian.[6]

1. The Lothrop is by far the simpler operation, as there is less surgery to be done, consequently less time and anesthesia consumed, less shock to the patient and only a moderate area of bone resection.

2. The eye and its adjuncts are not disturbed excepting for the cutting of the main return channels of blood from the upper lid, which will cause a moderate swelling and edema. In the Killian, superimposed upon the above trauma, is the more or less constant pressure upon the orbit, the length of time and degree depending upon the amount of bone resection required for the inferior wall, as well as the skill and dexterity of the assistant who is handling the retractors, the amount of edema of the eyelids, danger of paralysis of the upper lid, danger of injury to the eye and nerve being in direct proportion to the amount of pressure used and manner of handling of the retractors.

3. Danger of injury to the lacrimal duct with epiphora, marked in the Killian.

4. Continuation of discharge: In both the Lothrop and Killian, this is in all probability due to an infected cell that has been overlooked at the time of the operation. At the present

writing, I can recall four such cases. Three of these cleared up after a second operation; the first case being due to septal cell, the second and third to small supraorbital cells. In the fourth case, no secondary operation was done, as all symptoms excepting the discharge subsided.

In the Killian, a return of all the symptoms may occur, due to closure of the opening into the sinus by the sinking in of the soft parts and excessive granulation, caused by the removal of the upper portion of the lacrimal bone, nasal process and the adjacent frontal bone, which removes the bony support around the ostium.[7]

5. Fistula formation at the inner angle is more apt to occur in the Killian, especially so if instigated by the wick of gauze used for drainage. The removal of the bony support of the ascending process weakens this point, and if the drainage of the sinus thru the false ostium is occluded, it is very apt to result in a fistula.

6. Hemicranial anesthesia and neuralgia due to the cutting of the supraorbital nerve are the same in both operations.

7. Diplopia is never a cause for worry in the Lothrop, whereas it may be in the Killian, due to the dislocation of the pulley of the superior oblique.

8. Blindness on the operated side has been reported by several authors, one case occurring in our own clinic. This was probably due to injury of the optic nerve while curetting the sphenoid sinus. The optic nerve, like the facial, is very prone to insult, and it is quite possible that injury occurred thru a thin sphenoidal wall.

9. Death following frontal sinus operations, the shock of lowered resistance stimulating the growth of latent[9] infections in neighboring sinuses, which penetrate into the brain, the chief dangers being septic osteomyelitis or meningitis, and these are more prone to follow the Killian.

10. The shorter period of convalescence in the Lothrop, with a minimum disfigurement and the willingness of the patient to undergo the operation when it is known the eye is not to be disturbed.

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DISCUSSION.

Dr. Harris P. Mosher, Boston, Mass.: I wish I could share the author's feeling of satisfaction in the operative results which he obtains in operating on the frontal sinus, but I cannot. The frontal sinus operation in my hands and in the hands of men whom I have trained, is the most unsatisfactory of the operations which we have to do in connection with otolaryngology. I no longer do the full Killian operation. It is an operation of last resort in large sinuses; of first resort in small sinuses. I believe there are a number of good operative procedures on the frontal sinus, and each man will have to work out his procedure for himself.

One or two things I would like to take up as well as I can. The author said he had only four failures, but I do not know in how many cases.

I am not brave enough to use an electric burr. If anybody has the requisite courage and skill, I have nothing to say. I do not like to see anybody use it, but that is my personal preference.

A rubber tube as a drain to keep the newly formed nasal duct open—here again the writer is full of optimism. I cannot share it. I think the longer a rubber tube is left in the newly formed nasofrontal duct, the greater are the possibilities of trouble following. This operation is an attack from the air, i. e., from above. I prefer to attack the frontal sinus by frontal attack, that is, end-on in the old fashioned way, thru the anterior ethmoidal labyrinth.

As to the author's conclusions: The Lothrop is easier—yes. The eye is not disturbed—I agree. Injury to the lacrimal duct is marked in the Killian—I have never seen it, and the man who gets it needs further instruction in operative technic. Continuation of discharge in both the Lothrop and Killian is due to an infected cell that has been overlooked at the time of operation—I agree with that. Fistula formation at the inner angle is more apt to occur in the Killian—I have not seen it. Hemicranial anesthesia and neuralgia are the same in both operations—yes. Diplopia never a cause for worry—I have seen it. Blindness on the operated side—I have not seen it. Death following the operation I have seen. Shorter period of convalescence in the Lothrop—probably, yes. Those are my answers, my agreements and disagreements with his conclusions.

In my experience, the value of the modified Killian, like the Lynch operation, to which I am now turning for fear of osteomyelitis, in my hands is due to the removal of the whole of the floor, and to the discovery of frontal cells. My especial objection to the Lothrop, is that it is an attack from the air, an attack from above, and I do not see how you are going to remove the floor as thoroly and as easily as you can if you go in thru the old route, and I do not see how you can discover a frontoethmoidal cell in the majority of cases with this operation. By working backward from in front and from below, I have had more success in getting off the floor, and in finding a frontoethmoidal cell in series with the frontal sinus. I have lately looked up our frontal sinus results. Twenty-two per cent are a failure. Do not throw stones—you may live in glass houses. I have had a number of cases—I do not know the percentage—of permanent diplopia.

Dr. Ross H. Skillern, Philadelphia, Pa.: When one has under consideration an external operation on the frontal sinus, it is a pretty serious proposition. I look upon an operation of this character as essentially a life saving one—life saver in two ways, either actual or relative. By "actual" I mean where there is some meningeal orbital complication threatening, or some damming back of secretion, or fistula formation with fetid pus, which we know, if allowed to continue, will go on to very serious consequences and ultimately death. As a relative life saving operation, I would say where there is fetid pus and continual discharge, which despite every effort to terminate does not get any better, where tenderness continues over the frontal sinuses, perhaps getting larger in area, and in time showing a low grade osteomyelitis, where we have acute attacks much like appendicitis, growing worse all the time—it seems to me it may then be termed a relative life saving operation, which, if not done, will allow the condition to go on to something worse.

Now, suppose we have decided a radical operation is possible, what

are we going to do? Our first object is to cure the patient—if not 100 per cent, to cure him 90 or 80 per cent. Certainly 85 per cent is better than not at all. We are in there to do the best we can. We cannot all get 100 per cent successful operations, but we get as close to that as possible. Therefore, we have to do this radical operation. When we do make up our minds to do the operation, then we should do all that is necessary for permanent relief. I mean by that, that we should be just as radical as need be, taking out all the bone that is necessary, curetting away the diseased membrane, going down to the posterior wall if we have to, even to the dura; but we should also do the least that is necessary to bring about the desired result. In other words, let us not send a man to do a boy's work. If we have to do a certain amount, let us do that and no more.

Speaking of the operation itself, which my brother described, it seems to me that it has very distinct advantages over the Killian, that is, from our own personal experience. In the first place, it is attended by the least immediate danger—I mean meningitis, which we all fear as a horrible specter in the first 72 hours. After that time, we breathe a deep sigh of relief. Another immediate danger is the loss of or injury to the eye. Then comes shock. There is not a great deal of difference compared with the Killian, because all who have had experience with the Killian know that taking off the wall of the frontal sinus is one of the most difficult, dangerous and time consuming events of the whole procedure, but it also does away with excessive trauma and drainage. And it also affords relief from permanent deformity. There is no question about that.

The author did not go into one point clearly, and that is, where the periosteum and overlying muscles are sutured together, two sets of sutures are used; inside No. 1 catgut is used, and then those that bring the skin together are fine silkworm gut. In that way, instead of having everything opened down to the bone, as we do have when we make a cut clear thru, the periosteum grows together and the skin on the outside, thus doing away with a tremendous amount of scar formation.

In my experience, this method offers the best possibilities to the patient, the best hope for comparatively comfortable and rapid convalescence, with permanent relief of his symptoms.

Dr. Joseph C. Beck, Chicago, Ill.: This is no family affair with me, but it is an important question nevertheless, that after so many years of the radical treatment of the frontal sinus, from the time when Killian put forth a radical cure to this day, we should still discuss it. It shows plainly that we are having lots of failures in our work, and I can tell you that I have my share.

The modified Lothrop operation as Dr. Skillern, Jr. has described it, to my mind is of little use in the deep sinuses that require obliteration. If you obliterate such a deep cavity, you will get a deformity. In the smaller frontal sinuses, there is no such difficulty; it is in the large ones, that reach over the orbit and that recess backward and outward, that we get into difficulty. It was emphasized that the lining membrane would have to be entirely removed, in order not to get recurrence, and that is true, but what is going to line this large cavity?

We have recently heard of some research work under the direction of Dr. Mosher and it will be interesting to hear further reports on those

experiments. If it is as Skillern says, that we can expect a cavity to be made large by the removal of the nasal spine, as Lothrop advocates, and then have, so to speak, the bottom of the frontal sinus drop out, and then make a new relined cavity which will be ventilated and drained, I think we will have the ideal result.

On the occasion of my recent visit to Sweden, I saw the Barany operation on the frontal sinus, also cases that had previously been operated by him. The principle of the operation is the taking away of the bony floor of the frontal sinus, preserving the membrane, and establishing a newly lined channel by the removal of the anterior lateral part of the nose. He removes a part of the nasal bone, and then uses the mucous membrane from the anterior upper part of the nasal septum to reline the new channel, so that adhesions of the overlying skin cannot take place. He has a collapsing tube which allows drainage from the frontal sinus, but I do not see how it can be ventilated. I have seen frontal sinuses so diseased, that when opened, one knew that it was useless to leave a lot of membrane, because not only the membrane but the bone underlying it was diseased. In that case, the most radical operation will safeguard the patient from subsequent meningitis or brain abscess.

Dr. H. I. Lillie, Rochester, Minn.: The satisfactory management of suppurative frontal sinusitis may present great difficulties. The choice of operation for the relief of the symptoms will depend entirely upon the anatomic variation of the sinus as shown by roentgenographic studies, and the probable pathologic changes within the sinus.

It may be said in general, I believe, that there are two types of pathologic changes encountered within the sinus: those which respond to intranasal drainage, and those which require an external operation.

In 1921, I published a report of my experience with the two stage radical frontal sinus operation. Further experience with this method has been increasingly satisfactory.

If the external table is not diseased, the Lynch operation as the second stage has given satisfactory results. This operation, in my opinion, in certain cases is preferable to the Lothrop operation, because it exposes any frontoethmoidal cells to direct view.

If the Killian type of operation is necessary to remove the diseased external table, fear of considerable deformity need not deter the surgeon, for by bevelling the bone edges to avoid pockets and the abruptness of any depression, the final result may be quite satisfactory. The patient must decide whether or not he is willing to have some asymmetry of the forehead or continue to have the diseased condition.

Dr. Sam E. Roberts, Kansas City, Mo.: Some few years ago, I made the statement before this Academy that I considered there was no safe intranasal operation which included the rasping of the frontonasal duct; that the results were not sufficiently good to justify the procedure, either as a preliminary measure or as a measure attempting to cure. I believe the chief cause of failure is not the leaving of a few remnants of mucosa within the frontal sinus, but that the opening is not made large enough into the nose, and not enough of the floor is actually removed.

Regarding the use of a tube, I have been using that for ten years, since 1918. (See 1923 Transactions, page 322.) After the intersinus septum is removed, I insert a 19 or 20 catheter and allow it to remain for

six weeks. By leaving it for six weeks, there is formation of a ring of scar tissue which does not close.

Regarding a cure, there are very few sinuses that have been absolutely cured. I feel that if the patient is relieved of pain, even if he has a slight discharge, we may consider him cured.

Dr. William Mithoefer, Cincinnati, O.: It has been rightly said that with every change we progress, and I believe that Dr. Skillern has forged a new link in the chain of progress by modifying the Lothrop operation, making it less hazardous. I do not agree with him, however, in his method of approach. In the first place, I do not think it advisable to open the frontal sinus above the supraorbital ridge, because of the communication at this place of the diploic veins with the meninges. If we approach the frontal sinus from the floor, intracranial complications are less frequent.

Secondly, I do not think it advisable to curette the frontal sinus except when there is marked pathology, and even then not to curette, if possible, the entire cavity. We have all seen a maxillary sinus inflammation subside after drainage by means of a window resection. The frontal sinus properly drained will heal in the same way. What do we know concerning the character of the mucous membrane of the frontal sinus after operation? Are we going to have a mucous membrane as vital as the one we have removed? These are pertinent questions. I, therefore, believe that the frontal sinus mucous membrane should not be removed unless marked pathology is present.

And thirdly, I feel that another fault of the modified Lothrop operation is that we do not remove the laterally displaced ethmoid cells or the displaced inferior hiatus cells. In order to remove these cells, we must take away the lacrimal bone and also a large part of the ascending portion of the superior maxilla.

Postoperative retention is our greatest difficulty, and in order to overcome this complication, mucous membrane flaps are advisable. In a personal conversation with both Dr. Beck and Professor Barany this summer, I was told concerning Barany's method of using mucous membrane flaps. I have done this operation several times since my return. The mucous membrane flap is taken from the septum and brought up over the angle of the nasal bone. I have found the dissection of this flap rather difficult, but believe, that when a good flap is obtained, it is an ideal way of covering the entire wall of the frontal sinus opening on the orbital side. Another important factor in the frontal sinus operation is to cover the crista frontalis with mucous membrane. Barany does this by saving the mucous membrane of the frontal sinus, and makes a flap of the membrane to cover the crista frontalis.

We can all truthfully say that there is no ideal operation on the frontal sinus. In a series of 90 cases in our practice, we have not found it, but I think that if we keep on improving our technic we will finally be satisfied with our results.

Dr. Richard Waldapfel, Vienna: I must say I have not had any experience with the Lynch operation, but it might be of interest to say a word about our method of operation in Prof. Hajek's service in the Vienese Clinic, which has been developed in the last seven or eight

years, and has been published for the first time this year for the Copenhagen meeting. The salient point about this operation, as Dr. Beck and Dr. Mosher emphasized, is keeping the frontal duct open. We have followed some of our cases at the Clinic done by the Killian operation for seventeen years, and where the frontal duct was kept open, the results were good; where it was not kept open, the results were bad.

We make a small trephine opening over the frontal sinus, and then enlarge it as much as is necessary to permit inspection of the inside of the sinus. We then enlarge it downwards removing, if necessary, a piece of the frontal process of the maxillary bone, and the anterior ethmoid cells. We then remove the mucous membrane. Our aim in our further treatment is to keep this opening patent. We keep this fistula open for four to eight months, until we are absolutely sure that the cavity of the sinus is epithelialized and that there is no retention. We can thus control the whole course of healing, and are therefore able to be much more conservative in the removal of bony tissue than in the other methods. It takes a long time, it is true, and some people may think it too much to ask of a patient to keep it open so long But we prefer one operation and treatment of a little longer duration, to a shorter treatment with the risk of subsequently repeated operations.

We finally close the wound with one or two sutures, following which we often cannot tell where the patient has been operated on. Prof. Hajek says that the sole aim of all the different procedures thus far has been to obtain a good cosmetic effect, totally disregarding the functional side of the problem. Our primary object is a good functional result, and because we remove less bone in our operation than in the other methods we also get a better cosmetic result, despite the fact that it may at first appear to be disfiguring.

We have closely studied our operation for eight years, and have had such splendid results in a series of about forty cases that it has become the sole method of procedure at the Viennese clinic.

Dr. Fred Stauffer, Salt Lake City, Utah: Two years ago, I read a paper at the Colorado Meeting of this Society on the treatment of "Chronic Suppurative Frontal Sinusitis." One or two points were brought out in my paper that have not been mentioned here.

I wish to emphasize the importance of making skiagrams of every case before determining the kind of operation.

In the type of cases with broad sinuses, with many septa or ridges, it would be impractical to do anything short of a Killian operation, which aims at complete obliteration of the sinus by removing the anterior bony wall as well as the floor of the sinus, so that the soft parts above the supraorbital ridge drop back and adhere to posterior wall and the orbital contents come up from below, thus completely obliterating the sinus.

In these broad shallow sinuses, there is very little deformity following the Killian procedure, while in those cases in which skiagram shows the sinus to be deep antero-posteriorly, the Killian or any other operation which removes the anterior bony wall will cause a great deal of deformity.

These latter are cases very successfully treated by Barany or Lynch operation, which removes the floor of the sinus and establishes drainage from the sinuses into the nose.

Dr. Samuel R. Skillern, Jr., Philadelphia, Pa. (closing): Dr. Mosher said I had only four failures. He is wrong. I reported four failures that had a recurrence of the frontal sinus symptoms, but I had some deaths; I call those failures.

The rubber tube that Dr. Mosher does not approve of we leave in from ten days to two or three weeks to allow scar tissue to form around the tube, and in this way we hope to form a permanent outlet or ostium.

Answering Dr. Mosher and Dr. Lillie together, we do an intranasal operation first with the hope of cure, and if we get that, of course we do not do any more operating.

We have had a fistula in a half dozen Killian operations.

Dr. Beck, we do not attempt to fill the sinus. We bring the periosteum over and make the sinuses as near normal as possible, the periosteum being the outer wall. In making your perforation thru the septum, you must remember that you have a normal ostium on the other side that has not been touched. You have an ostium on the well side that may give sufficient aeration for both sinuses, with drainage thru the newly enlarged ostium of the infected side.

Dr. Mithoefer spoke of the diploic veins. By taking off the anterior plate, you leave the diploic veins open to infection to the meninges. The inner plate, as you know, is of ivory like consistency and contains the diploic veins. We have never had them carry back the infection. We curette everything we find diseased.

Dr. Mosher and Dr. Mithoefer spoke of the ethmoidal cells. Can you tell whether there is an ethmoidal cell between the roof of the orbit and the floor of the frontal sinus? You know how far down the frontal sinus goes, and how far up the roof of the orbit comes, and if there is more than one-sixteenth inch of space between them, you may be pretty sure you have an ethmoidal cell present. Suppose you have an ethmoidal cell that is diseased, the bone forming the roof of this cell is weaker, and if you take a rasp or chisel and touch it lightly, you may break thru into the orbit, but what harm have you done? Not a particle. If you go into the orbit far, all well and good; if you go into the supraorbital cell and find disease and curette it out and get drainage, you will cure your patient.

SYSTEMIC INFECTION FOLLOWING ANTRUM LAVAGE.

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For many years, it was our custom to irrigate the antrum in acute and chronic disease of this cavity, without taking the temperature before or after the lavage. It happened that on many occasions, the patients informed us that several hours after the lavage, they had a severe chill, followed by fever. Visiting these patients at their homes, it was found that there was present a marked reaction in the nose with an associated nasopharyngitis, and sometimes a tonsillitis, which had not been present on the day when lavage of the antrum took place. In many instances, with the patient bedfast, we again proceeded with daily antrum lavage, thinking because of the large amount of pus present in the antrum at each washing, that this was the logical procedure. We soon learned, however, that an acutely, and also that very often a chronically, inflamed antrum should not be irrigated if there is present a fever prior to, or after the lavage, and that to puncture the antrum at this time, is to invite trouble and complications.

Patients are very often able to care for the infection in an antrum provided no other irritant appears upon the scene, such as the irritation which is caused by an antrum lavage. As soon as another insult is added to the infection already present in the antrum, an imbalance of the body mechanism takes place, the bacteria gain the upper hand, and many times, because of the injury to the mucous membrane from puncture, a secondary inflammation ensues, and a local or general sepsis may be the result. It is far better judgment, in our estimation, to abstain from irrigating acutely inflamed antra in the presence of fever, and furthermore, to desist in our efforts, if after irrigation, there is a rise in temperature. This applies to chronically, as well as acutely inflamed antra.

It is true, there are many patients with antrum disease in whom there never occurs a reaction after irrigation. This im-

munity is to be explained, first, by the constitutional idiosyncrasy of the individual, and secondly, by the character of the bacteria. An individual reacts against certain irritations according to the character of his inner qualities, and these inner qualities we term "constitution". A strong constitution means immunity and sufficient defensive action of the cellular elements to prevent an imbalance from taking place. A weak constitution, from whatever cause, readily becomes affected by an added irritant, whether it be bacterial, atmospheric, psychic, or mechanical. The bacteria themselves may be of a nonvirulent type, until an outside irritant, in the form of lavage, enters their midst. At such a time, there may be but a mild reaction, which may be looked upon as a beneficial one; but it is otherwise if the patient is unable to meet the attack. It is then that an imbalance results, with fever and the possibility of systemic involvement.

What means have we at our command to prevent a sepsis following an antrum lavage? In the first place, the antrum should never be irrigated in the presence of fever. A mild degree of fever, not exceeding 100°F. is no contraindication, provided the reaction be carefully watched. If there is a distinct rise of temperature following the lavage, we must abstain from further irrigations until the fever has subsided. At all events, the temperature should be taken before, and also in the evening of the day of the lavage. If chills and fever have followed the lavage, there should be no attempt made to repeat the same on the following day. We must, furthermore, proceed with caution, if on the day following the antrum lavage, there is present a fibrinous deposit in the region of the puncture. This is a local reaction, which may be followed by systemic invasion if further irrigation is continued.

It is undoubtedly true, that the majority of cases of acute maxillary sinus inflammation heal with little or no treatment of the nose or sinus itself. It has been our experience to see many antra return to normal, without further treatment, in patients who had suffered a severe reaction after the first lavage. It is our custom never to irrigate the cavity, if reaction has occurred, until at least a week or more has elapsed after the disappearance of the fever. It is astonishing how often the antrum at this time is found free from secretion, and the skiagram has returned to a normal appearance. This observation has been the means of making us more conservative regarding

the immediate performance of lavage of the antrum in acute cases.

Our records reveal the interesting fact, that many of our patients suffering with acute disease of the maxillary sinus, have had a mild reaction following lavage. These patients made an uninterrupted recovery without further lavage of the antrum during the active stage of the disease. We have also seen patients with chronic disease of the maxillary sinus who had a rise of temperature after lavage, and are of the opinion that in such instances, it is far better to abstain from irrigating the cavity and to proceed a short time after the reaction has subsided with either dental or maxillary sinus surgery.

We have observed five very severe forms of infection following lavage of an acutely inflamed antrum. In the one instance, twenty-four hours following the puncture of the antrum under the inferior turbinate, the patient developed a very severe form of erysipelas, which spread rapidly over the entire face, and which caused him to be bedfast for almost four weeks. The antrum of this patient was never irrigated again, and some time later, the skiagram showed that the antrum was clear. Another patient, eight hours after antrum lavage, had a severe chill followed by a temperature of 103°F. Examination on the following day revealed a septic nasopharyngitis and a beginning tonsillitis. There was also a marked local reaction in the nose, with a fibrinous deposit in the region of the puncture under the inferior turbinate. This patient was also bedfast for many weeks because of a complicating nephritis.

It is true that greater reaction follows puncture of the antrum thru the bony wall of the inferior meatus with a sharp canula. The use of sharp instruments in this region may readily injure the plexus of veins which are usually well marked on the lateral wall of the nose, along the course of the lacrimal canal. Furthermore, a sharp pointed trocar may be more apt to injure the engorged veins present in the thickened mucosa lining the diseased antrum cavity. Injury to the veins not only makes it possible for air embolism to follow the puncture, but also for the development of a septic thrombophlebitis with consequent pyemia. We prefer to irrigate the antrum thru the middle meatal wall, and always use a blunt pointed canula. Many years ago, Sandman demonstrated the fact that the anterior and posterior ends of the inferior turbinates have a greater susceptibility to stimulation than any other part of the

nose. We will, therefore, because of the normal sensitiveness in this area, have a greater reaction following antrum puncture under the inferior turbinate. There is, furthermore, less likelihood of injury to the venous plexus if the cavity of the antrum is entered in the region of the middle meatus with a blunt pointed canula, on account of the extreme thinness of the wall in this region, and the fact that very often puncture is found unnecessary because of the presence of normal or accessory ostia.

Injury to the mucous membrane and to the veins as the result of puncture, plays but a small part in the causation of subsequent local and systemic reaction. We feel confident that this is true, in view of the fact that many times, in irrigating thru the middle meatal wall, we have been able to enter the maxillary sinus thru a normal or accessory ostium without injuring the membrane, and after this procedure, there has frequently occurred a reaction with chills and fever. There are, therefore, other factors, both constitutional and local, that are the cause for the severe reaction. One of our patients, with dental empyema of the antrum, had a temperature of 103° F. for two days, following an irrigation thru the normal orifice. Irrigations in this case were not resumed until after the extraction of the tooth, at which time there was no rise of temperature following the procedure.

A recent case of acute maxillary sinusitis had a severe reaction, which lasted five days and was associated with a marked albuminuria. The most severe reaction that we have seen occurred in a patient with acute bilateral maxillary sinusitis, who at the time of the first lavage also had a septic nasopharyngitis. She was irrigated on four successive days. After each irrigation, there was a chill and rise of temperature, at one time reaching 104° F. Following the last irrigation, the patient had metastatic processes in her joints, with marked cervical adenitis on both sides. A week later, she developed a pleurisy with effusion on the left side, and a pyelitis. The pyemia continued for four weeks. The precarious condition of this patient during several weeks of her illness, gave us great anxiety. It appeared for a few days as if there might be a fatal ending. This case taught us an important lesson, and since that time, we have been more conservative in our treatment of acute antrum disease.

Killian, Hajeck, Schlittler, and Halle have reported cases of sepsis as a result of antrum lavage. Most of the patients recov-

ered, with the exception of the one case reported by Halle, in which death resulted. Riedel, in 1925, reported six cases of sepsis after lavage of the antrum, with one death. Scheibe also reports two cases, with one death.

We are firmly convinced that antrum lavage is a procedure not free from danger of subsequent systemic infection. We, therefore, wish to impress upon you the importance of taking the temperature before and after irrigation, and not to continue irrigation if there is present fever exceeding 100° F. In these cases, we are confronted with a greater danger after irrigation than we are without irrigation. In order, however, to prevent the establishment of a latent antrum disease, it is far better to irrigate the antrum after the temperature has been normal for at least a week. We have found that many of these patients who had a large amount of pus present at the first washing, and in whom, because of a reaction following the lavage, irrigation was abstained from, had no pus in the irrigating fluid several weeks after the temperature had been normal. We must conclude from this, that many cases of acute antrum disease get well without irrigation of the cavity.

DISCUSSION.

Dr. William V. Mullin, Cleveland, O.: Dr. Mithoefer has presented an important surgical principle so completely that I can add practically nothing. Perhaps it is because I agree with him so thoroly, that I have not been so unfortunate, or I might say unlucky, as to have seen any of the systemic infections that he has reported. I have made the statement before, that pus in the antrum is not the worst feature of an infection. It may quite take care of itself. It is the edema and the infection within the membrane that gives systemic absorption. I have many pathologic slides showing an abscess in the membrane completely surrounded by edematous tissue. Should I have an acutely infected maxillary sinus that after careful deliberation I felt needed surgical interference, I should, with the slightest trauma and manipulation possible, open it in the canine fossa, leave it open, and do nothing further until later.

I have been unfortunate enough to have had at different times one disagreeable complication following puncture of the antrum, that is emphysema of the orbit.

In 1914, while spending some time in the Killian Clinic, I began washing the antrum thru the natural ostium or else thru the middle meatus. Now I confine myself to that method entirely, and I have not punctured thru the inferior meatus in over two years. With a little practice and patience, it will be surprising how often a blunt cannula can be passed thru the natural slit. Should this not be located, only the slightest amount of pressure is required to go thru. This method has

several advantages. It is humane, and therefore gratifying to the patient. Because of this, it will be done sooner and more often than if inferior meatus puncture had to be resorted to. It is to be noted that doctors, as a rule, refuse to have the antrum punctured.

Dr. Frank B. Kistner, Portland, Ore.: Anyone who has seen the severe and sometimes terrific septic reaction that occasionally follows antrum lavage will appreciate the importance of the subject which Dr. Mithoefer has offered for discussion.

Dr. Mithoefer's advice about puuncture and lavage in acute and subacute antrum infection is good, but I feel that he does not put it strongly enough. Treatment at this time should be the same as that for acute and subacute infection in any other part of the body. Absolute rest in bed and the use of such physiotherapy as will promote the comfort of the patient and help shorten the duration of the infection will greatly decrease the number of cases in which it will be advisable to resort to lavage. To my mind, instrumental manipulation in an acutely or subacutely inflamed nose is contraindicated, and the gratifying result occasionally following lavage during these stages of inflammation is no justification for the unnecessary risk.

In the presence of any elevation of temperature or any evidence of acute or subacute infection in the nose or throat, the question of puncture and lavage should be given very careful consideration.

When lavage seems advisable, it is our rule not to repeat sooner than 48 hours. As Dr. Mithoefer has said, we have no way of knowing the susceptibility of the individual or the virulence of the infection he may harbor. One of the most severe cases of sepsis I have seen occurred 8 hours after a diagnostic puncture in a patient who had shown no evidence of sinus activity for 3 months, and showed nothing in the wash to indicate activity. He was in a serious condition with chills and high temperature for a week, and totally incapacitated for 3 weeks.

In the case of acute empyema, when pain and fever are not controlled by rest and the usual measure for relief of acute infection, I think it is much safer and gives quicker relief to drain thru an opening in the canine fossa, rather than to attempt puncture and lavage or any other instrumentation in the presence of the acutely inflamed and engorged mucous membrane of the nasal cavity. Drainage in this way is continuous rather than intermittent, as in lavage by puncture, and we escape the danger of repeated traumatism in a septic field.

I do not agree with Dr. Mithoefer as to the advisability of irrigation thru the middle meatus with a blunt cannula. Unless the middle turbinate has been removed, I think there must be more traumatism from this procedure than from a careful puncture with a sharp trocar in the inferior meatus. It is our routine procedure. In our clinic, the average number of antrum punctures has been over one hundred per month for the last three years, with eight septic reactions. No fatalities. One developed mastoiditis. Eighty per cent of the punctures were done for diagnosis only, and the septic reactions, all but one, occurred in cases under treatment. Previous to this time, with a smaller number of patients, our septic complications were much more frequent.

I have for long felt that treatment of an infected antrum by puncture and lavage is practiced too much. Most of the acute cases will recover

without this treatment, and in the chronic cases, we have not eliminated the infection, but have induced a latent stage that will become active again at the least provocation.

Cultures from infected antra in over four hundred cases operated in our clinic have shown one or more types of streptococci in 95 per cent. It is incredible to think that any surgical procedure, no matter how insignificant it may be, can be carried out with impunity in the presence of this type of infection.

Dr. Howard V. Dutrow, Dayton, O.: My object in discussing this paper is to emphasize, by a case report, some of the danger signals and pitfalls pointed out by the essayist. Dr. Mithoefer's paper is timely, in view of the fact that there is an increasing number of serious reactions and deaths following a simple maxillary sinus lavage. A few years ago, we were concerned with the discussion of what seemed to be a mechanical cause of death, during or immediately after puncture and irrigation of the maxillary sinus. Unfortunate experiences from this cause seem to have diminished or disappeared entirely, and we are now confronted with the serious reactions due to infection, both active and latent. Any assistance which we may derive from the consideration of this serious complication in a meeting of this kind, should interest us as rhinologists.

The following is a brief report of a fatal case occurring in my private practice in May, 1928:

A. McC., age 47, an athlete and judge on the bench of the Common Pleas Court. History of general malaise and neuritis in right arm. Diagnosis: Maxillary sinusitis, right, subacute. No elevation of temperature. Operation: At eleven o'clock Saturday morning, under cocain-adrenalin anesthesia, the anterior tip of the inferior turbinate was elevated with a Killian nasal speculum, and an opening made thru the nasoantral wall in the inferior meatus, with a Thornwald drill, which was enlarged with a cutting forceps to permit the easy passage of a Killian irrigating canula. The lavage was made with a saturated solution of biborate of soda. The solution was returned cloudy with many muddy mucopurulent clumps, characteristic of an old streptococcus infection. He felt no discomfort until eleven o'clock the next day (Sunday), when he had a slight chill, and one hour later he had a temperature of 104° Fahrenheit. A few hours later, the right side of his face began to swell, and by evening his right eye was closed. The next morning (Monday), the temperature was 102.4° Fahrenheit and the pulse 120 and not of good volume. The swelling had extended to the forehead and scalp and down the side of the neck. There were a few blebs on the skin at the outer canthus of the right eye. Cultures from these blebs revealed a pure culture of streptococci. The skin was red and elevated at the margins of the swelling, resembling erysipelas. At noon, there was total suppression of urine, the radial pulse was scarcely perceptible, and the swelling had extended to the crown of the head and downward over the anterior surface of the chest. During the afternoon, there was pulmonary edema, and death ensued at eight P. M., just thirty-three hours after the initial chill and fifty-seven hours after the sinus lavage. Hurried consultations and the administration of antistreptococcic serum and so forth were of no avail. One is struck by the rapidly fatal course of the general systemic infection

in this case, and our utter inability to cope with the swift and tragic fury of an overwhelming toxemia. Dr. Mithoefer is forunate in that all of his cases recovered. Dr. Ross H. Skillern, in a personal communication, stated that he recently had a similar case, which was desperately ill for a period of from four to six weeks.

Many deaths, as all of you know, from general sepsis following the extraction of teeth, are being reported to the literature at the present time.

Most sinus operations are elective, thus permitting the selection of a favorable time, without the apparent presence of local inflammatory reactions or fever, for the operation. But when we do operate, we are compelled to enter an almost completely closed cavity, harboring in more than 90 per cent of instances that deadly organism, the streptococcus. I do not believe it makes much difference whether we irrigate thru an opening in the middle or inferior meatus, provided the opening is of sufficient size to permit ventilation and drainage. My experience in entering the maxillary sinus in the middle meatus has been one of great difficulty, probably from lack of skill, which has caused me to favor the opening in the inferior meatus.

The time and frequency of the irrigation, I believe, should be governed by the individual requirements of the case and the surgical judgment of the surgeon.

In views of this and other case reports, we may clearly visualize what may happen to any of us engaged in this work, at any time. If by reporting this sad experience, I may aid a colleague in avoiding a similar one, I shall feel that I have done my duty, at least towards the members of our chosen specialty.

Dr. Robert Sonnenschein, Chicago, Ill.: My remarks will be confined to a discussion of irrigation of the maxillary sinus by way of the middle meatus. A good many years ago, Dr. Pierce of Chicago washed out my antrum this way, and this procedure has been carried out hundreds of times since. The experience was not unpleasant, was devoid of much difficulty, and since that time, in our office, we routinely irrigate the antrum this way. It is quite true, that once in a while it is impossible to reach the natural or accessory opening because of marked deflection of the septum, but with this exception, we have been able to enter the antrum, if not via one of the openings, then by a puncture thru the thin membranous wall of the middle meatus.

The danger of penetrating the orbital cavity, I think, is very slight if you have an X-ray picture and know the size of the antra. One occasionally finds cases in which the pus is so thick and tenacious, that irrigation by way of the middle meatus will not permit sufficient pressure to flush it out. In view of the fact that many men, including ourselves, report that this method is devoid of pain, shock, and usually of bleeding, we firmly believe it is the method of choice in most cases.

Dr. William Mithoefer, Cincinnati, O., (closing): My object in presenting this paper was to give a word of caution to all of us who are in the habit of daily irrigating the antrum without giving due consideration to the patient himself. There are many individuals coming to the office of the rhinologist who have a slight fever, and who are having a

streptococcic hemolytic infection of the nasal accessory sinuses. If the temperature is not taken before lavage, we are likely to produce a systemic invasion following the procedure.

One of the speakers said that he did not approve of washing thru the middle meatal wall. I want to say a few words in regard to the technic which is used for this purpose. A Ritter sound is first used, in order to become acquainted with the topography of the part. It is very impractical, in my opinion, to irrigate the antrum thru a puncture made under the inferior turbinate, when there is already present an accessory opening which is easy of access. If no accessory or normal ostium is found with the Ritter sound, gentle pressure is made against the membranous wall, and the same pierced. A von Eichen's canula is next introduced at the spot where the wall was punctured, keeping the blunt pointed end of the canula directed downward.

I do not think Dr. Dutrow's case was one of systemic infection following lavage, but rather one which followed operation on the antrum. I, furthermore, do not think any operative procedure should be done in the presence of an acute inflammation of the nasal mucosa, without first making a bacteriologic examination in order to see whether you have present a hemolytic streptococcic infection.

ULTRAVIOLET LIGHT AS AN AID IN THE PREVENTION AND TREATMENT OF INFECTIONS OF THE UPPER RESPIRATORY TRACT, WITH CASE REPORTS.

MILLARD F. ARBUCKLE, M.D.[1]

ST. LOUIS, MO.

The invitation to speak on this subject was accepted with misgiving, because I felt the lack of broad experience with the remedy mentioned in the title. My remarks will be confined chiefly to personal observations, with such references as it has been possible for me to include. This is a subject which has aroused unusual interest in the minds of the medical profession and the laity within recent years.

In looking over the literature, it is found that opinions are numerous regarding the value of ultraviolet light, the indications for its use and the contraindications and dangers attendant thereupon. Many are enthusiastically in favor of its use, while others are correspondingly pessimistic regarding its value.[2, 3]

Ultraviolet light as a remedy, and the apparatus for supplying and applying it, have been widely and persistently advertised by certain manufacturing concerns, with proclamations that seem exaggerated to say the least, and I think it highly probable that for this reason alone many doctors have refused to have anything to do with this remedy either as a prophylactic or a curative agent.

Commenting on the sale of ultraviolet generators directly to the public, the Council on Physical Therapy reported as follows:[4]

"The Council on Physical Therapy of the American Medical Association, on the basis of the present available evidence, is convinced that the sale of generators of ultraviolet energy to the public for self treatment is without justification. The Council bases its condemnation of the sale of such apparatus for this purpose on the following grounds:

1. The uninformed public could not take the proper precautions in administering treatments, and as a result, severe general burns or grave injury to the eyes might ensue.

2. Those not familiar with the possibilities of such apparatus would be led to place unwarranted confidence in the therapeutic value of such treatment by the claims that might be made in the literature advertising such generators, and to undertake to treat serious conditions not amenable to such treatment.

3. The unrestricted possession of such therapeutic means would tend to deprive people of expert diagnosis by encouraging them to make self diagnosis.

4. Such practice would encourage the sale of useless and fraudulent lamps which would be advertised as generators of ultraviolet rays, since the public would have no means at its disposal to determnie the quality or quantity of the radiant energy emitted by such lamps.

For the foregoing reasons, the Council on Physical Therapy considers as detrimental to public welfare the sale or the advertising for sale, directly to the public, of a generator of ultraviolet energy. Under rule 11 of its Official Rules, the Council will declare inadmissible for inclusion in its list of accepted devices for physical therapy, apparatus manufactured by a firm whose policy is in this matter detrimental to public welfare[8]."

My interest in the matter of prophylaxis and treatment of disease with this agent was stimulated by articles appearing from time to time which seemed to contain evidence worth while considering.

In order to gain first hand information, I had installed in my office two and one-half years ago a water cooled lamp and an Alpine Sun Lamp. During this period, I have treated about 100 cases in which ultraviolet light was used as an adjunct. This series consisted chiefly of cases of chronic sinusitis, with a few cases of furunculosis of the external ear and a few cases of acute tonsillitis.

During the early part of this period, I used the quartz rod applicators intranasally in about 25 cases. Some of these had been operated and some had not been operated.

There was apparently so little benefit obtained by this method, that it was discontinued. The same statement holds true in the cases of furunculosis and acute tonsillitis.

In the remaining nasal cases of the series, the results which apparently were obtained by the use of ultraviolet light were of sufficient value to justify reporting.

The type of case deriving the greatest benefit in my experience has been the child or adult, more often the former, wh

had a chronic infection in the paranasal sinuses with frequent exacerbations, accompanied by secondary anemia, lack of appetite, daily low grade fever, lymphadenitis, probably an unexplained cough and a condition generally below par. It is not infrequent that patients with this sort of history and findings are given a diagnosis of tuberculosis and treated accordingly.

I have had great difficulty in the past in getting some of these patients to improve, especially during the winter months.

It appears to me that many of these have shown surprising and unexpected improvement in all phases of their disability promptly after the addition of the ultraviolet light to their treatment.

The effect first to be noticed was improvement in the appetite and a spirit of well being. The local evidence of infection was decreased and the periods between acute exacerbation were lengthened.

It was found that short exposures one minute up to five, of the entire body at a distance of 30 inches, daily or on alternate days, was more satisfactory than larger doses.

This is in line with the experience of Maughan and Smiley[5] as reported in their splendid article in the Journal of Preventative Medicine, January, 1928. They refer to the work reported by Barenberg, Freeman and Green[6] published in the J. A. M. A., 1926, in which it was found that in infants irradiated in an attempt to improve the general health negative results were obtained with regard to the prevention of colds. Improvement was noted during the first month, but during the second, third or fourth months they were more susceptible to colds and other infections, and in other ways were less normal than their controls.

In this series of cases (Barenberg, Freeman and Green) large and frequent dosage was given, so that the skin assumed a muddy brown color.

In the same article, McKenzie and King,[7] as a result of investigation made in their medical practice in England, state that "the common cold should be treated by ultraviolet light irradiation. Local and general irradiations are used and results warrant the statement that this will be the method in the future in dealing with this very British disease."

I have had a few cases in which exposure to the light during the acute stage with fever was followed by unpleasant symptoms such as increase in fever and prostration. This has caused me to refrain from its use under these conditions.

While I am convinced that an important biochemical reaction takes place within the body when exposed to ultraviolet light, I have refrained from trying to discuss this feature, preferring to leave that part of the discussion to those with a more intimate knowledge of chemistry and physics.

I have had no experience with diathermy.

As a result of my experience with ultraviolet light therapy and that of some of my friends, I am convinced it is a remedy which helps many individuals but that it is not free of danger.

I have to date not had any accident or any untoward result worthy of mention, but I have seen cases elsewhere in which the reaction was so marked as to amount to a severe burn over a considerable area of skin. I saw one individual (not my patient) who had a severe burn of the soft palate and pharynx. In two cases which were seen in consultation, an early but unsuspected pulmonary tuberculosis apparently suffered marked reaction, with serious increase of all the symptoms.

This remedy should, therefore, be used only by those who are fully conversant with the indications and the contraindications for its use, and who are equipped to apply it safely.

Case No. I: D. M. Age 7 years. Pt. came in with a retropharyngeal abscess in November, 1927. Altho her tonsils and adenoids had been removed about a year before she had failed to grow. The abscess was incised and she was given exposure to the ultraviolet light at weekly intervals for about two months. During this time, she gained nine and one-half pounds.

Case No. II: T. V. S. Age 32 years. Feb. 12, 1920. This is one of my old patients, who has had much trouble with sinusitis and toxic labryrinthitis. He has been in fairly good condition for several years. Within the few months before November, 1927, he had repeated attacks of acute exacerbation of an old sinus infection, with marked increase in dizziness, lack of appetite and loss of weight. On November 28, 1927, he was placed on irradiations, with almost immediate improvement. His gain in weight was very rapid, as well as the gain in his general condition. His exacerbations have been much less frequent and less severe since his exposure to the ultraviolet light.

The above mentioned cases are fairly typical of the progress of many of this type of case exposed to the light.

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DISCUSSION.

Dr. McKim Marriott, St. Louis, Mo.: There is nothing new under the sun, even the therapeutic qualities of sunlight, for that has been recognized ever since the dawn of medicine. It has only been recently, however, that the action of the sun's rays on the human body has been accurately studied, and then it became known that only a very small portion of the solar spectrum was capable of exerting a physiologic effect. These particular rays can be produced artificially and in a degree of intensity considerably greater than that of sunlight, and the physiologic action is essentially the same. The most important effects may be briefly summarized:

There is a local reaction of the skin followed by pigmentation, the absorption into the blood of partially altered body proteins leading to a nonspecific protein reaction, a bactericidal effect which is present only at the surface, an absorption of radiant energy by the blood, so that this radiant energy is carried to distant parts of the body. The effect of the absorbed radiant energy is to promote calcification both in rickets and tuberculosis, to stimulate the formation of blood platelets, and in cases of anemia, to stimulate more rapid regeneration of blood. Whether or not irradiation with ultraviolet light increases resistance to infection is a disputed point. There is a popular idea that it does. Scientific proof of this is slight. Animal experimentation shows irradiated animals to be more resistent to typhoid vaccine. The bactericidal power of blood is decreased by irradiation, however. The opsonic index of the blood is not changed by irradiation of the body. The resistance of normal animals has, at times, been increased by exposure to ultraviolet light, but in the presence of infections, the resistance has been decreased by the same exposure. Irradiation in small amounts has been shown to increase the complement of the blood; in large amounts, to decrease it. Experimental work on children, carried out in a large clinic, showed that children irradiated daily by a mercury vapor lamp gained in weight no more rapidly than those of a control series. They had no higher red blood count, no higher hemoglobin, and suffered from no fewer rhinopharyngeal infections during the course of a winter. They all appeared healthier, however, because of a coat of tan. That they were actually healthier, could not be demonstrated.

That individuals exposed to sunlight out of doors are less likely to suffer from infections, that their resistance to tuberculosis is increased, and that their nutrition is improved, is an accepted fact. These good effects are not observed, and certain harmful effects are observed, when the exposure to sunlight is excessive. Some of the good effects are

undoubtedly due to the fresh air and stimulation of the skin rather than to the ultraviolet light alone, for these effects can not be duplicated by exposure to artificial sources of ultraviolet light indoors. Ultraviolet light is capable of doing harm. It is a two edged sword. There is a popular wave of enthusiasm at the present time for all forms of light therapy, and this type of therapy is recommended for almost any condition to which the flesh is heir. Its limitations are now beginning to be realized. Careful scientific observation is essential. The subject should be approached in an unprejudiced manner, and data collected and carefully analyzed. The essayist has done this. His observations are presented for what they are worth, and because of the carefulness with which he has made them and the unprejudiced spirit in which they have been presented, they are worth a great deal.

Dr. Harry L. Pollock, Chicago, Ill.: The paper, as you noticed, was not published in the Presessional Volume, and I did not have an opportunity of seeing it until a day or two ago. I had prepared a discussion on physical therapy in otolaryngology, but the essayist limited his paper to the upper respiratory tract, and I do not think there is much to be said. In a paper read before this Academy three years ago, I stated that all of these physiotherapy agents had a limited field, and if their employment was restricted to this field, they would give good results. In our experience, the use of ultraviolet rays in infections of the upper respiratory tract and sinuses gave absolutely no result. Its use was equally valueless in cases of acute sinus disease. There are conditions in which we have obtained excellent results in the use of this adjunct, but since they were not included in the subject of the paper discussed, I shall refrain from mentioning them. Very good results have been obtained in cases of small children, in whom the tonsils and adenoids had been removed, brought to us with a diagnosis of chronic bronchitis. Some of these cases improve rapidly with small daily doses. They lose their cough, gain in weight, and their general well being is much enhanced.

The Doctor stated that he had had no experience with diathermy. We have employed it in many conditions, and feel that all of these energies, ultraviolet, infrared light, faradic, galvanic, etc., fill a need in the treatment of certain conditions in our special field.

In my opinion, they should be included in every ear, nose and throat man's armamentarium. They are limited in their use, altho, as the Doctor has said, the manufacturers claim that they are a cure for all the ills that human flesh is heir to. They are, however, a valuable adjunct in ear, nose and throat practice. We have had excellent results from these agencies in various conditions, but since the paper refers only to sinus disease, I have nothing further to add.

Dr. T. S. Blakesley, Kansas City, Mo.: I can agree with Dr. Arbuckle from the standpoint of the results from body radiation in these children. There is no question about the results one will obtain with the ultraviolet light used on the body.

In a general way, I have noticed two distinct effects from ultraviolet light. We have been using it now for about four years. The first definite effect is that it is a germicide. Organisms exposed to the light cannot live for more than a few seconds. That is demonstrated very beautifully in the throat in Vincent's angina. If one cleans off the slough from an

area of Vincent's and exposes it to the quartz light for a minute or two, and then searches for the organism the following day, one will find they have practically disappeared and the Vincent's will recover.

Second, chronic inflammations about the eye are practically all benefited by exposure to the ultraviolet, until an acute reaction is produced on top of the chronic condition. The result is marked, especially in chronic conjunctivitis of long standing. One produces an acute inflammatory reaction which lasts four or five days, and when that subsides, the chronic condition goes with it.

The operations Dr. Arbuckle speaks of are not necessary. Each has its virtues and its faults, and the faults are all eradicated in case the operations are not over used.

As to the question of whether the light is harmful to the lens of the eye, we have used it in therapeutic doses and produced quite marked reactions in the eye, but we have yet to see any bad effects on the lens. I believe the ultraviolet light is absorbed before it gets to the lens. In those cases that are exposed to the arc light, it may possibly be due to the actinic rays.

Hay fever is benefited by the quartz light, particularly if there is long enough exposure. It desensitizes the nasal mucosa. Exposure every other day, for three or four treatments, usually makes the patient comfortable. It is not a cure, but it does help. In the sphenoid and antrum, those cases that have postoperative discharge, we have found it possible to light up an acute reaction persisting for perhaps a week, by use of the quartz light (by means of quartz rods), and with the subsidence of the acute reaction, the chronic condition was benefited.

With diathermy, one has two things to consider. First, its use as a treatment. We have found it beneficial in acute sinus conditions to relieve the pain, and we have yet to see any bad systemic effects from its use there. In sinuses that are full of mucopurulent secretion, the secretion is thinned, permitting its escape. Of course, we always follow such treatment by shrinking methods with cocain, pituitrin, or ephedrin, and usually get good drainage.

The other use of diathermy is electrocoagulation. It is especially valuable in such conditions as angioma of the tongue. One can coagulate the mass under local anesthesia, and when it sloughs out, one will have a smooth scar. This is also true in operations on cancer about the face. One gets destruction of tissue in a bloodless field.

Dr. J. L. Myers, Kansas City, Mo.: I want to thank Dr. Arbuckle for giving us a paper on "ultraviolet light." However, you notice in the Program that the Committee requested a paper on Physical Therapy in Ophthalmology and Otolaryngology. We wanted that subject brought before our Academy, because every one of us has had numbers of patients who say that they have used some kind of physical therapy. On making inquiry, in some cases, we learn that some such modality has been used. Often, when the facts are obtained, it is discovered they are using a small instrument sold by electric light companies, to which a vacuum tube is attached which gives a violet color as the current passes thru the tube. This they call a violet ray. It is neither a violet ray nor an ultraviolet. The using of this instrument is very much like using patent medicine,

which is condemned by all physicians. This instrument also should be condemned.

One reason we wanted a paper on physical therapy, was because we wanted our members to have some knowledge of various modalities used in physical therapy, and to know where they should use them and where not. It is not possible to learn all of this merely in one paper, but such a paper would direct one in such a way as to guide him both in purchasing instruments and in studying the uses of these modalities.

There is no doubt that diathermy is the best method for administering heat deep in the tissue; that coagulation of certain neoplasms is as good, if not the best way to remove these growths; that air cooled ultraviolet radiation is helpful in many conditions; that water cooled ultraviolet is beneficial in others. All are adjuncts in therapy, none of them, nor are all of them, a cure all.

What is needed is a working knowledge of each modality. It is just as necessary for one to study these modalities before he can intelligently use them as it is that he study medicine before using drugs scientifically. When he has learned the physiologic action of each, he then knows where it is indicated. Moreover, he will find it is as necessary to be exact in the use of each modality as it is necessary to be exact in using medicine.

One drawback in the advancement of physical therapy today is the absurd statements made by salesmen as to "great cures" secured by using various modalities. I know men who have purchased all kinds of instruments because some salesman has assured them that they would do great things with them. These physicians have not learned how to use this new paraphernalia or in what conditions the various modalities are needed, and the result has been that they have been disappointed and have condemned physical therapy.

If there is anything that requires knowledge and exactness, it is physical therapy. One must make his diagnosis as carefully as when he gives medicine or uses surgery, and then he must know the mechanism of his modality and remember that all modalities change when used. Don't leave all to a technician all the time, but check up your machines and see if they are up in efficiency as they should be. There are some things we should not do and some we should do, to get good results in physical therapy:

1. Don't depend on salesmen's talk alone—investigate.
2. Don't expect a small instrument, because it costs less, to do what a larger and higher voltage instrument will do.
3. Don't depend on your technician entirely.
4. Do become familiar with the various modalities of physical therapy.
5. Do continue making exact diagnoses.
6. Do use the correct modalities correctly in cases indicated.

If these things are done, one will find physical therapy a helpful adjunct in his therapeutic armamentarium.

DR. J. L. SHELLMAN, St. Paul, Minn.: If you are in possession of a Kromayer water cooled lamp, there is one condition for which you will find it almost a specific. That is a strong statement, but I am amazed at the results I obtain in eczema of the external canal or the orbit. If you use the Kromayer lamp for a minute with a No. 9 rod in the ex-

ternal canal, then take off the small filter and give the full effect of the rays for a minute or a minute and a half, I think you will be agreeably surprised at the results.

About sixteen months ago I was in Vienna and was invited by a doctor to go with him to some small cities, where he was going to do some operations on children. I went as assistant and spectator, and I could not help notice the color of the children that were brought in to be operated on. I naturally asked what gave them that peculiar color, and the doctor told me it was his regular treatment to have these children come in for exposure to Alpine sunlight a certain number of minutes every week. If you have ever been in that section, you know that in February, March and April there is very little sunlight and a great deal of rain, and the results they obtain from the use of this light, I can say from personal observation, are very satisfactory.

Dr. M. F. Arbuckle, St. Louis, Mo., (closing): Within the last year or so I have been using the socalled infrared heat in a certain number of cases of subacute and acute sinusitis with severe pain, and I have been surprised and greatly pleased at the results obtained in some of these cases. I had one case in which I could not stop the pain by any other treatment I knew, but which was relieved by exposure to infrared heat.

I do not do eye work, but I recall a case of keratitis and iritis which was seen in consultation, in which the eye lesion resisted to an unusual degree. After general irradiation with the ultraviolet light, the eye appeared to heal almost over night.

Regarding hay fever and desensitization of the nose by ultraviolet light, I cannot understand how it works. My conception of hay fever is that it is the result of a condition we call allergy, and if it is an allergic condition, how can we relieve it by exposure to ultraviolet light? That is more than I can understand at the present time.

I am very much obliged to Dr. Myers for his remarks regarding the matter of finding out what we are doing before we start. I am in fear and trembling all the time of what may happen. I have not lived up to all his specifications, but I do as well as I can.

I am reminded by Dr. Shellman's remarks regarding eczema that I did try it on two or three cases of eczema of the external auditory meatus, but I am sorry to say that my results were not as satisfactory as his.

DISEASES OF THE LINGUAL TONSILS.*

HAL FOSTER, M.D., F.A.C.S.

KANSAS CITY, MO.

Years ago, Morell Mackenzie and Lennox Browne of England, called attention to the diseases of the lingual tonsils. They were soon followed by Austrian laryngologists, also by Knight, Bosworth, Lefferts and Ingals in the United States. In recent years, some of the members of this Academy have written upon this subject. Notwithstanding all the above articles, the subject remains interesting, both on account of its frequency and the seeming neglect it receives at the hands of many physicians in daily practice.

During the recent winter, I saw quite a number of patients suffering from lingual tonsil disease. Several of them had been treated a long time and were not benefited. They had taken large quantities of cough syrups. They were very nervous, unable to sleep at night, due to the distressing tickling sensation in the throat, constant desire to swallow and irritable cough. One of them was expectorating blood stained mucus. The expectoration of blood had brought on a general depression, caused by fear of something dreadful just ahead. They were nervous from loss of sleep, due to tickling in the throat and a constant desire to cough and swallow.

After seeing these patients relieved and restored to their normal state of mind and health, I thought it would not be out of place or time to call attention anew to this all important, but seemingly neglected subject.

The lingual tonsils are located on the base of the tongue, just in front of the epiglottis, consisting of lymphoid tissue. This bilateral mass of tissue is irregular and frequently well developed in adults. It will often be found to continue laterally with the lower part of the faucial tonsils.

The physician may see here all the diseases affecting the faucial tonsils, namely lingual varix or varicose veins, abscess

*With Lantern Slides.

or lingual quinsy. Blood may be seen escaping from these varicose veins by the use of a laryngeal mirror. They may be the seat of focal infection. I have seen several cases of lingual quinsy. This kind of abscess will be found just underneath the capsule. They should be immediately incised and contents thoroly evacuated under local anesthesia. In disease of this region, one will have to treat the entire glossoepiglottic region when found to be envolved. Tubercular and specific ulcers are occasionally found here, also carcinoma.

Etiology: Some individuals seem prone to have varicose veins. Intemperance in eating, drinking, smoking, repeated attacks of cold and chronic infected faucial tonsils are prominent factors in causing disease of the lingual tonsils.

Pathologic Conditions Noted: Bleeding from the varicose veins on the surface of the tongue, acute and chronic inflammation, hypertrophy from the size of a pea to a walnut, inflammation following influenza, measles and scarlet fever. I have seen keratosis located in this region; varicose veins are also seen here. The veins may be of a bluish or a very red color. Vincent's angina may be also located here, when the faucial tonsils are affected. The most frequent affection is hypertrophy, especially in middle life. Both men and women are afflicted but it seems to occur more frequently in men. Inflammations following streptococci infection are often seen in the lingual tonsils.

Method of examination: The lingual tonsils can be seen by the use of the laryngeal mirror and head light.

Symptoms: A constant dry cough. Empty swallowing. Sensation of a foreign body in the throat. A tickling sensation at night, which prevents sleep and causes these individuals to be very nervous. I have seen several cases where the lingual tonsils were so large as to press on the epiglottis, causing some interference with breathing. Lingual quinsy will at times cause some respiratory disturbances. In the great majority of cases, the disturbances will be local and harmless as far as life is concerned. The loss of sleep and the sight of expectorated blood will cause these patients to be nervous and unfit for their daily vocations, brought on from fear. As you well know, fear is a mighty factor in causing mental depression in our patients. It may be difficult for the patient to correctly localize the seat of the trouble. The distressing symptoms vary at different times, marked with greater intensity at night. While eating, the discomfort disappears. All symptoms are more marked in neurotic subjects. On examination with the laryngeal mirror, the area between the epiglottis and the base of the tongue will be found a reddish like tumor, consisting of lymphoid tissue, hypertrophied in size from a buckshot to a marble or olive. Some of these individuals will tell you when the laryngeal applicator touches the location of distress.

Diagnosis: Made easily by means of the laryngeal mirror and a good reflected light. Hemorrhage from the throat is of course not always from the lingual tonsils. It may be due to pulmonary tuberculosis. Bleeding from the lingual tonsils can be seen by the laryngeal mirror, and the source of the blood demonstrated. The sputum should always be examined for tubercle bacilli, and a careful chest examination made. Rales are absent in lingual tonsil disease.

Prognosis: Aside from the great local and mental discomfort produced by lingual tonsil disease, this condition yields quickly to the proper treatment and leaves no unpleasant sequel in its train. The lingual tonsils should be examined in all undiagnosed coughs.

Treatment, Medical and Surgical: The treatment should be used, which the laryngologist's experience has taught him offers the best results and least discomfort to his patient. The medical treatment consists of topical applications of astringents, nitrat of silver, tr. iron in glycerin, iodin in glycerin. Caustics such as

chromic and trichloracetic acid, can be used to advantage. In applying any of the above named drugs, great care should be used not to permit the drug used to spread all over the pharynx, but confined strictly to the area or spot desired to be treated. The mild cases will be relieved quickly by applying weak solutions of iodin in iron and glycerin, using great care not to use in such large quantity as to run all over the base of the tongue, but confined strictly to the place desired to be treated. Solutions of cocain should be applied before using any severe measures. In my hands nothing compares with the use of the galvanocautery at a red heat. Cocain or butyn can be applied locally for anesthetic purposes. The long curved galvanocautery electrode will give splendid results. After the cautery is used, there is not very much pain. The after treatment consists in using cracked ice in the mouth, spraying the throat with menthol in benzoinol. Orthoform lozenges will control the pain if used immediately before eating. Hot antiseptic gargles should be used several times a day. If the galvonacautery electrode is used at a red heat there will be no after bleeding. Anesthesin-calcidin troches dissolved slowly in the mouth every three or four hours will make the patient much more comfortable. Lozenges made of anesthesin-acetanilid in cocoa paste repeated every four hours will afford great relief after the cautery has been used. The lingual tonsillotome can be used if the surgeon desires, also the snare with a curved canula. One should use the local anesthetic of choice in removing the lingual tonsils. The writer has relieved large numbers of patients of this annoying and distressing condition, along with the troublesome hacking cough and hemorrhage and the fear that always goes with lingual tonsil disease.

In all undiagnosed coughs, look to the lingual tonsils; by doing so you will make many grateful patients, who will be your friends while life lasts. We should never forget in laryngology, frequently it is the small ailment corrected that benefits our patients and establishes a lasting confidence in the medical profession that nothing else can do. Happy individuals, grateful and benefited patients are the physician's greatest friends. This we should strive to bring about along the journey of life.

References.

Lennox Browne, Morell Mackenzie, Kyle, Tilley, Ballenger, Hall, Gleason, Barnes, Bruck, Moore, Bosworth, Dan Mackenzie, Ingals, Packard, Gile, Shurly, Grayson, Coakley, Phillips, Knight, Sir St. Clair Thomson, Sir Logan Turner and Barnhill.

DISCUSSION.

Dr. James J. King, New York City: I have occasionally found lingual tonsil disease where I thought it played some part as a focal infection in systemic disease. I have occasionally seen abscesses at the base of the tongue, as Dr. Foster has stated. I have also seen these troublesome coughs simulating tuberculosis due to this disease. I think the most troublesome case of lingual tonsil disease I have ever seen, was where the base of the tongue was the seat of mycosis, where the patches had to be picked off one at a time, and I had trouble in eradicating the disease because it was not entirely removed when I operated on this patient.

In treating these cases, I also agree with Dr. Foster. I think perhaps the cautery, or diathermy, is the best way to treat them. In that way, you obviate the probability of somewhat serious hemorrhage, which is sometimes possible. I have had no experience with serious hemorrhage of this kind, but some men have had very troublesome hemorrhages following lingual tonsil operation.

At the present time, and for some time past, I have been removing the lingual tonsil at the same time I did the operation for the faucial tonsil. I do this in the ordinary way with a snare and hook. Miles has perfected a very good instrument for the removal of these lingual tonsils, called the Miles tonsillotome. But I would utter a word of caution in cutting the whole surface across the base of the tonsil, for fear of producing serious contraction of the tongue which would be annoying to the patient. In other words, instead of cutting the whole surface, take out alternate bits, so you do not get so much contraction. I think that is a valuable point. Also, at the base of the tonsil, be careful not to have the cut surface of the faucial tonsil overlap the cut surface at the base of the tongue, since if you do, you are apt to get a severe contraction of which the patient will complain. I have had that happen in a number of cases, and I have remedied it by following the Yankauer method of massaging the point with the index finger once a day and having the patient do that, gradually causing a stretching until the contraction is overcome.

I think it is especially urgent in a case of tuberculosis, where there is a positive sputum, to clean up the base of the tongue, and in those cases I would recommend a cautery at red heat, so as not to have any hemorrhage at all. Hemorrhage is to be avoided in these tubercular patients, as it frightens them as well as causes loss of blood which is undesirable.

Dr. E. E. McKeown, Denver, Colo.: This lymphoid tissue covers well the posterior third of the tongue, laterally almost touching or connecting with the palatine fossa tonsils, and from there down to the epiglottis. This gives a rather large area for foci of infection. The follicles run anterior-posterior or parallel to the length of the tongue, are rather shallow in comparison to the crypts found in the pharyngeal tonsil, but due to this shallowness, the crypts are larger and therefore subject to more direct infection; but on the other hand, the shallowness does not allow material to be retained in these crypts for any length of time. It would seem, therefore, that the amount of infection derived from this lymphoid tissue is not as much as is found in other portions of Waldeyer's ring.

Infection of the lingual tonsil is decidedly more frequent in adults,

especially so in the female. This is usually between the ages of thirty-five and fifty. Of course, the symptoms depend on the size of the nodules, and even on these nodules touching some other portion of the throat, such as the epiglottis. This gives rise to the sensation of an irritation such as a foreign body may cause, resulting in a cough. The retention of foreign material is not so likely, therefore, not so frequently the cause of retention deeper in the structure of the tongue or lymphatic circulation. Abscesses occur occasionally beneath the capsule which separates the lingual tonsil from the deeper structures of the tongue. The hypertrophied lingual crypt indicates increased activity, and this may be physiologic. If so, should surgical interference be instituted? Unless some pathologic condition is present, the hypertrophied lingual tonsil may be only a local manifestation of some general condition.

The fine meshwork of lymph vessels found in the mucosa of the lateral lymph tissues of the faucial tonsils is not present in the lingual. The method of treatment of the lingual tonsil is not on trial, but rather the judgment of the throat specialist. The lingual glands beneath the mucous membrane or capsule of the lingual tonsil are especially abundant around the foramen cecum, to a less degree around the vallate and foliate papillae. The posterior surface of the tongue drains into the internal jugular group, or lateral collecting group of lymph glands. To my mind, it is very essential in doing the operation not to cut too deep into the structure, but rather to remove the lymphoid tissue immediately above the capsule, thus causing very much less scar tissue. This scarring may be so severe or extensive as to cause a pulling by contraction of the tongue, thus narrowing the arch. One case in mind came in for a frequency in choking, that is, food going into the larynx, and inability to properly control swallowing. The only abnormality found, was that where the lingual tonsils had been removed, there was a smooth, shiny, scarred surface. I believe the sensation had been so impaired as to allow the food to slide over this without the normal feeling. I am wondering also if some of these operations are not performed during the acute lingual tonsillitis attacks. Do we have any more reason for removing this acutely inflamed tissue than in the acutely inflamed faucial tonsil? Cutting deep into the tissue is apt to give us an excessive amount of scarring.

Dr. George B. Jobson, Franklin, Pa.: There is no doubt that pathologic conditions in the lingual tonsil occur more frequently than we are aware of. This is probably due to the fact that our examinations are superficial, and in some cases, this region is entirely neglected. Here we have a mass of lymphoid tissue which is practically devoid of capsule except a very thin membrane, and it is surrounded with a profuse blood and lymph supply which exposes it to contamination or infection.

I wish to cite a case of severe infection showing how dangerous infection may be in this particular region. The patient was brought in to me, after ten days illness, by the family physician, who gave the following history. A minister, 50 years of age, on December 2, 1925, developed a sore throat which centered at the base of the tongue. The postpharyngeal wall was normal, as were the faucial tonsils and palate. Temperature slightly elevated and accompanied by slight chilliness. The second day, his sore throat increased and there was swelling at the base of the tongue, but the rest of the throat was normal. Temperature at

this time was 101°; pulse 100. He complained of a stiffness of the throat muscles and considerable chilling. The condition went from bad to worse, until at the end of ten days, there was an immense swelling at the base of the tongue which prevented his swallowing. Temperature 103°, pulse 110. A large abscess ruptured at the base of the tongue, and for three or four hours he was considerably relieved. In the middle of the afternoon of the next day, sloughing occurred, with severe hemorrhage, and this continued intermittently until the following day, when he was brought to the hospital almost exsanguinated and very septic. I found, on examining the throat, that there was a large ragged ulcer just to the left of the lingual tonsil and some blood oozing from this region, which was controlled by pressure. The neck was badly swollen, and there was a certain amount of crepitus, either from gas in the tissues or from air being forced in while swallowing. I inserted a pair of hemostats thru a slit in the skin, and on spreading them, got only blood and some bubbles of air. The patient died three hours afterward from sepsis and general depletion. This case to my mind shows that we must treat this region with a great deal of respect.

Dr. Harry L. Pollock, Chicago, Ill.: There are two points not touched upon by the essayist, which I would like to bring to your notice. We all know, that in children who have had their tonsils and adenoids removed early in life, there is a tendency on the part of Nature to cause a compensatory hypertrophy of lymphoid tissue, which often takes place in the lingual tonsil. Certainly, many cases presenting symptoms due to lingual tonsil hypertrophy or lingual tonsilar disease give a history of removal of tonsils and adenoids early in life.

A symptom of this disease which I have found in a limited number of cases is a persistent burning of the tongue, especially toward the tip. These cases yield to the treatment as outlined by the essayist. Another condition which we find in the lingual tonsil is leptothrix, which is always secondary to leptothrix of the faucial tonsil. The primary condition is often very annoying, and the only treatment for the faucial disease is tonsillectomy.

Generally speaking, the treatment for hypertrophy of the lingual tonsil is cauterization, either by actual thermal cautery or by chemical cauterization. Deep X-ray therapy applied thru the side of the neck is often of great benefit, as we know that X-rays have a selective, destructive action on lymphoid tissue.

We see a great many cases of dry cough, as described by the Doctor, relieved by two or three applications of a weak solution of silver nitrat, after they have failed to respond to treatment by internists or other specialists. Some of these persistent coughs are present in cases of but slight hypertrophy; it does not require a tremendous hypertrophy to cause these symptoms.

This is a very timely presentation, and I agree with the essayist, that a more careful investigation of the lingual tonsil in these cases will be conducive to a great deal of good to our patients as well as to ourselves.

Dr. Millard F. Arbuckle, St. Louis, Mo.: This is a condition which is frequently present and often overlooked. I would like to quote what

Dr. Sluder told me years ago, namely, that he thought it was one of the plagues of humanity—chronic lingual tonsillitis.

The cause I have come to believe in many cases, if not the majority of cases, is a chronic suppurative sinusitis, with a constant flow of infection over the lingual tonsil. I have seen or heard of a few cases in which very distressing conditions arose after the removal of the lingual tonsil by any method, X-ray or otherwise, namely, the destruction of a so-called aberrant thyroid, which happened to be the only thyroid the patient had, and after the lingual tonsil had been removed, the symptoms of hypothyroidism occurred. I have seen a few cases in which the lingual tonsil so closely resembled malignant disease in appearance, that it was necessary to do a biopsy before the question could be decided. I have also seen a few cases of lingual quinsy with edema of the glottis so severe as to threaten the patient's life.

The operation of choice for lingual tonsillectomy, I think, is the dull cautery, but if the cautery is allowed to cut deeply, I am always afraid of secondary hemorrhage. Dr. Sluder told me he nearly lost his life when he was a young man from a deep cut thru the lingual tonsil.

It seems to me the thing to do for these cases is local application to the lingual tonsil, and careful treatment of the nasal infection which nearly always exists along with it.

Dr. William Mithoefer, Cincinnati, O.: We are all probably in agreement with the thoughts expressed in the paper, but I rise to ask a question of Dr. Foster, and that is, how many of these patients upon whom he operated were not relieved? This brings up the question of differential diagnosis in all cases where there is present a chronic focus of infection at the base of the tongue. We make it a point never to consider the lingual tonsil as a causative factor without first examining the floor of the mouth. The glands of the floor of the mouth very often become enlarged and painful when the focus of infection is in the tonsils, teeth, or nasal accessory sinuses. These glands very often remain enlarged after tonsillectomy, and give rise to discomfort and pain in the pharynx simulating very much lingual tonsillar hypertrophy.

Glands on the floor of the mouth are best examined by means of bimanual palpatation. Two fingers of one hand are placed on the floor of the mouth, and the fingers of the other hand placed on the outside, at the angle of the jaw, so that the glands, especially the submaxillary and small lymphatic glands that lie posterior to the submaxillary, may be palpated. Very often, on palpation, the patient will say that the symptoms are aggravated, but after several minutes of massage a relief of symptoms will frequently be noticed. If you do find a condition such as this, it is poor judgment, in my estimation, to do any work on the lingual tonsil without first using a systematic course of massage of the glands on the floor of the mouth. Relief of symptoms is often obtained in this way, without resorting to treatment of the lingual tonsil.

Dr. E. Lee Myers, St. Louis, Mo.: Dr. Armin Gundelach of St. Louis some years ago called attention to the relation of the lingual tonsil to diseases of the thyroid. Since that time, I have been very much interested in the role that the lingual tonsil plays in its relationship to pharyngeal surgery.

I will diverge somewhat from the subject and ask, should the lingual hypertrophy which is sometimes seen at the time of the tonsil operation be removed? There are many cases in which the tonsil operation seems to increase lymphoid overgrowth, not only in the region of the lingual tonsil, but also in the faucial cavity. Is not this lingual hypertrophy a reflection of the patient's lymphoid diathesis? Many of these cases associated with lymphoid overgrowth do not seem to be improved following the tonsil operation. In my opinion, the lingual tonsillar hypertrophy should be handled less with surgery and more symtomatically, i. e., applications of silver nitrat, cautious removal piecemeal, or the judicious application of galvanocautery.

Dr. Hal Foster, Kansas City, Mo., (closing): I thank the gentlemene very much for the part they have taken in the discussion. I see I was not wrong in thinking this is an important subject. Some of the older writers spoke of this as globus hystericus.

The reason I use red heat in the cautery is that it sears the parts, and they do not bleed. I have had hemorrhage from white heat, and in the cases Dr. Arbuckle mentioned, I think the cautery was not hot enough. If you use white heat, you certainly will have hemorrhage. Lymphoid tissue bleeds very easily. I have been called to stop hemorrhage from tonsils on which general practitioners had operated, who could not stop the hemorrhage. I usually find a piece of lymphoid tissue low down on the tongue, and then the trouble starts with hemorrhage. Usually, when that is taken out, the hemorrhage stops. But by always using red heat, you will have no trouble with hemorrhage.

There are always different methods of operating, always have been and always will be. I have seen these cases where the cough stumped the best internists. They are not familiar with looking down behind the tongue. They say nothing is the matter with the chest. The point I want to make is, do not overlook a cough.

If I was operated on, myself, for faucial tonsils, I would not want the lingual tonsils interfered with at that time. Usually, when patients come to see you, they want relief right away. They have been coughing and are nervous, and they want relief. That is what they are looking for, and they want it immediately. Some slight treatment, as I have outlined in the paper, relieves them of the cough.

Once I was called to testify in court, where a general surgeon undertook to operate on a child about twelve years old and gave him a general anesthetic. The child choked to death from abscess. I did not see the patient, but I was glad to help this man out and make the best showing I could. The mistake he made was giving the child a general anesthetic. A laryngologist would have used a local anesthetic and held the child's head over to let the pus run out.

Someone asked if all these cases were relieved. I have never seen one that was not relieved. Sometimes they recur when they have a cold. I had one come back recently, but two or three treatments stopped the cough and relieved the trouble.

I think it would be much better to leave the faucial tonsils alone, and make two operations of it. One of the best articles I have seen recently was written by Sir St. Clair Thomson in his new book on this subject.

AIDS FOR THE HARD OF HEARING.

HORACE NEWHART, M.D.

MINNEAPOLIS, MINN.

The important service now rendered the hard of hearing by many recently improved aids, and the relatively meager amount of literature on the subject now available, justify a paper of the above title. Fairness to the various manufacturers obviously precludes the specific mention of names. The most important articles dealing with the subject have been published during the past year as the direct result of the interest and activities of the deafened themselves, represented by their national organization, the American Federation of Organizations for the Hard of Hearing. The articles worthy of special mention include that portion of the report of the Committee on Scientific Research relating to "A Method for Rating the Performance of Hearing Aids", of which Dr. Harvey Fletcher is Chairman, and the Report of the Committee to "Survey Instrumental Aids to Hearing", Dr. Douglas Macfarlan, Chairman. Both reports are published in the Proceedings of the American Federation of Organizations for the Hard of Hearing for 1927. The work of the latter committee was carried on with the generous co-operation of the United States Bureau of Standards. A further recent contribution by Dr. Fletcher is entitled "Instrumental Problems of the Deaf", presented before the conference on the Problems of the Deaf called by Dr. Knight Dunlap, Chairman of the Section on Anthropology and Psychology of the National Research Council, Washington, D. C. January 20th and 21st, 1928.

Mechanical aids to hearing are divided into two groups, the nonelectric and the electric.

To the former class belong the many varied types of horns, conches, speaking tubes, auricles, etc., of different materials with which the past generation was more especially familiar. They depend for their effectiveness on the collection and amplification thru resonance of the sounds of speech, so that they are

conveyed to the ear in intensified form with the least possible distortion. Some instruments of this type are used to transmit the sounds by bone conduction thru the teeth or cranial bones. Many individuals with a moderate hearing loss find this type serviceable and inexpensive. The chief objection is founded on their conspicuousness, weight and unwieldiness. According to Miss Annetta W. Peck of the New York League for the Hard of Hearing, whose experience with aids to the hard of hearing is most extensive, many more people should be encouraged to use the nonelectric aids earlier and more persistently than is now the case.

Electric aids to hearing have been extensively used only during the past twenty-five years. It should be recalled in passing, that it was due to the efforts of Professor Alexander Graham Bell to produce an electric aid for his hard of hearing fiancee, later his wife, that the world today enjoys the telephone.

For practical purposes, in connection with the use of electric hearing aids, Fletcher groups the hard of hearing into four classes: (1) Those who readily hear the ordinary conversation voice at two to four feet and who show with the audiometer a loss of less than thirty sensation units in the better ear thruout the range of the human voice, i. e., from one hundred to three thousand double vibrations per second. (2) Those whose loss is from thirty to sixty sensation units for the same pitch range. (3) Those with a loss of from sixty to eighty sensation units. (4) Those whose loss is greater than eighty sensation units. He states that if electric amplification could be made "ideally free from distortions", the needs of the first three groups could be met by two instruments, the first sufficiently powerful to overcome a hearing loss of thirty sensation units, equivalent to a magnification of one thousand times, the second having a power sufficient to overcome a loss of fifty sensation units, representing a magnification of one hundred thousand times. The first group require no special aid for interpreting conversation at close range, and in an ordinary room with the usual amount of noise, such a person should hear up to a distance of ten feet with the aid of the weaker instrument, for a person speaking in such an environment instinctively increases the intensity of his voice to overcome the handicap produced for normal individuals by the noises present. In a quiet place, however, a person of the first group would need an instrument of the weaker type for distant conversation, that is with the speaker further

away than ten feet. A person belonging to group two would require the less powerful instruments for close work. For distant hearing, it would be necessary to place the transmitter near the speaker or to resort to the more powerful instrument. The requirements of a person in group three would be met only by the more powerful instrument. In practice these various requirements are largely met by giving increased intensity, by adding more transmitters and otherwise increasing the volume of output thru the employment of resonating bodies. All types of instruments are fitted with controlling devices, permitting the selection of all intensities below the maximum at the will of the user. Added power is secured in some types by substituting for the "midget" receiver a larger one used with the headband.

Persons of group four present peculiar problems and require instruments of special construction, with the disadvantage of increased distortion and lessened portability. Nevertheless, instruments of this type have been recently perfected with vacuum tubes and improved transmitters, and are more efficient in volume and relative freedom from distortion. The larger models are not portable. Equipped with multiple outlets having individual control and fitted with phonograph attachment, they are most useful aids to the teacher in schools for the deaf, helping to get over to those with but a small remnant of hearing, rythm, accent and pitch.

The nonportable type is also used most successfully in the home or place of business. With its aid, the ordinary telephone is easily used. It should be stated that our telephone companies are now prepared to supply, on a rental basis, special equipment for ampliying the sounds of the receiver so that those with a considerable hearing loss can now use the telephone with satisfaction.

In a practical way, we may properly ask what should be the duty and attitude of the otologist with respect to aids for the hard of hearing? He should obviously first of all inform himself regarding the possibilities and limitations of the means now available to help the deafened. He may no longer be able to improve his patient's hearing by treatment. Too often, he can not even hope to successfully conserve what hearing remains. Nevertheless, the patient rightly looks to the medical man as the one person peculiarly able to understand his special needs. He expects of the latter sympathy, guidance and such

advice as will help him most effectively to keep in touch with the world, which he feels is gradually receding from him.

In performing this function, the physician should urgently advise two things, which tho of the greatest importance, are often neglected or postponed, to the great disadvantage of the patient. These are, first, to encourage the person who has a hearing loss sufficient to be a handicap, with the prospect of further loss, to at once procure the best hearing device he can afford; and, second, to begin seriously the study of lipreading, or speech reading, the greatest of all boons to the deafened.

The tendency to regard these measures as something to be employed only as a last resort, is to be vigorously condemned as fallacious and pernicious. If a person delays adopting these aids until he is severely deafened, he loses much valuable time, during which he unnecessarily burdens himself with added exhausting effort, and afflicts his friends. If he resorts to the instrumental aid late, it requires a far greater effort to reeducate his ears. He finds special difficulty in excluding or ignoring all the adventitious sounds which the normal person from lifelong habit unconsciously suppresses. The adventitious sounds are all necessarily amplified equally with the useful sounds of speech, and constitute the greatest obstacle for one beginning the use of an electric aid. Therefore, it is most important to encourage every beginner to persist in his attempts to use the instrument over a considerable period, until he acquires sufficient familiarity and skill to make the advantages outweigh the disadvantages. Then he finds real happiness in his new accomplishment.

The great advantage of proficiency in lipreading as a supplement to an instrumental aid is not adequately appreciated. Even persons with normal hearing rely unconsciously upon lipreading in their interpretation of ordinary speech. To the hard of hearing, the need of this supplementary aid is emphatically greater. In fact it is indispensable. For those with a severe hearing loss, even with the best hearing aid, many of the consonants and diphthongs are not perceived, tho the vowels are still heard. Lipreading enables such persons to supply the unheard elements of speech. Accordingly, the otologist should insist on the early study of lip reading as soon as he discovers in his patient a permanent or threatened handicap.

Instruction in lip reading is now advocated for such school children as are found on audiometric test to have a hearing

defect sufficient to be a possible factor in causing retardation in their work. The repeater in school is abhorred alike by the educator and the tax payer. Some of our cities go so far as to provide thru the public schools instruction in lip reading for the adult hard of hearing.

Of very great assistance to the hard of hearing, and for that matter to the rest of us who are not so afflicted, would be the promotion of a national movement to encourage the proper enunciation and pronunciation of the English language. This point is so obvious as to require no further elucidation, tho in our daily contacts we are apt to lose sight of its importance.

Previous to the efforts of the American Federation of Organizations for the Hard of Hearing to investigate hearing aids, one occasionally heard various criticisms and even complaints regarding the instruments or the methods employed in selling them. These related, in some instances, to extravagant or unwarranted claims by some manufacturers or distributors for the performance of their products or to their methods of selling them. Other complaints were in the nature of protests against the expense of maintenance, largely because of the employment of special types of batteries. Many felt that the charges for a trial rental were unjust.

These causes for criticism so far as they existed have largely been corrected, especially by the larger manufacturers, who are now rendering a highly ethical and efficient service. The mechanical features of many instruments have lately been greatly improved. The newer midget receivers are remarkable in lightness, compactness and inconspicuousness, the latter quality making an especial appeal to the many who seem never to overcome their sensitiveness to their handicap. Some manufacturers employ standard battery units such as are ordinarily used in flashlights, which are inexpensive and are everywhere procurable.

The otologist is often asked regarding the cost of an electric hearing aid. Some makes put out a simple, efficient outfit for twenty-five dollars, which is useful, however, only for those with a relatively small hearing loss. In general, the cost is proportionate to the amplification. Several makers produce excellent instruments, with a wide range of power, and with various accessories in the form of different receivers and containers for the amplifiers, which serve as resonators, for from sixty-five to one hundred dollars. Instruments for the very hard of hearing employing vacuum tubes are proportionately more expensive,

the nonportable types for home and office use costing several hundred dollars. Those for school use are even more expensive.

A greatly appreciated service now provided by several local organizations for the hard of hearing, is the maintenance in their quarters of a permanent exhibit of various types of hearing aids, which may be inspected and tried out by prospective users. Those in charge have no commercial connection with the manufacturers. In some cases, the social worker endeavors to procure for the needy, discarded or abandoned hearing aids at a minimum cost, or gratuitously.

Since different persons have very different requirements, which can be best met only by one of many hearing aids, it is evident that the selection of a device which will give the greatest satisfaction is a very personal matter. Final purchase should be made only after a thoro trial, for which the manufacturers make liberal provisions. Determination of the type to be tried out will be greatly facilitated by a careful otologic examination, which in our experience is made more valuable by the audiometer test. An audiogram is very useful if the patient can not go personally to the distributor, and is helpful in watching the general progress of the case.

Statements have been made, leading to the inference that the use of electric hearing aids has a curative effect. Every otologist should vigorously combat such claims, whether made directly or by implication, for they are calculated to abuse the credulity and ignorance of the afflicted. To the physician it must be clear that no hearing device can remove the pathologic causes of deafness. We admit that in many cases there is an improvement in the ability to hear speech, following the use of a hearing aid. This, however, is the result of the aquisition of greater skill in the interpretation of speech sounds. Actual increase in acuity for pure tones as the result of using a hearing device, so far as we know, has not been scientifically demonstrated.

On the other hand, subjecting the auditory apparatus, whether it is normal or has suffered material impairment, to loud sounds for a prolonged period in the hope of improving the hearing, is to be condemned as harmful. Hence the danger of too great amplification.

It should be emphatically stated that the otologist's duty to his hard of hearing patient does not cease when he has prescribed a hearing aid and lip reading. Too often, however, the patient is permitted to gain the impression that he no longer

needs otologic care. On the contrary, so long as he has remaining a useful remnant of hearing acuity, he should at least be under the observation of an interested medical man, so that no possible factor may be overlooked which might lead to greater hearing loss. The deafened person, more than all others, needs to conserve his hearing. He should have a careful examination including an audiometric test at stated intervals. He should at all times be guarded from the deleterious influences of excesses in food, tobacco and alcohol. He should avoid the harmful effects of improper elimination, inflammatory disturbances of the upper respiratory tract, including acute and chronic sinus disease, focal infections, wherever located, and severe fatigue. Such a program can be best directed only by the otologist, to the end that the patient may retain the highest possible hearing efficiency.

Among the most important problems which confront those who would effectively help the hard of hearing individual is the matter of his psychology. It is perhaps the greatest obstacle to his success and enjoyment of life. The average deafened person by virtue of his impairment has lost confidence in himself. He soon develops an inferiority complex, which grows upon him thru the isolation with which he seeks to conceal his handicap or to avoid being constantly reminded of it. This situation is benig met most efficiently thru the activities of the local organizations for the hard of hearing which now exist in many of our cities. No one understands better than the deafened themselves the needs of one with acquired deafness, and the otologist who will induce his hard of hearing patient to associate himself early with a group of this kind is rendering a distinctive service. The achievements in the way of rehabilitation of the hard of hearing person thru these agencies are in many instances most surprising.

Thus safeguarded, and fortified with the mastery of a properly selected instrumental aid, having acquired his maximum of skill in lip reading, the average hard of hearing person has conquered to a great extent his handicap, and should courageously take his share of the responsibilities and joys of life.

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DISCUSSION.

DR. MAX A. GOLDSTEIN, St. Louis, Mo.: The few remarks that I shall make on this very timely paper are based in substance on one statement in the paper: "No hearing device can remove the pathologic causes of deafness." I was standing in the back of the room with one of our St. Louis colleagues and I said, "What a wonderful thing radio amplification is for a large group like this," and he replied, "Yes, but to a certain extent it changes the quality of the voice, and to a certain extent impairs the personal equation of the speaker." That is true about every electric hearing device. The two hearing devices which are of use to the deafened individual are the electric and the nonelectric. All the electric devises are based on the radio and the telephone principles, and there is no doubt that the deafened person who uses an electric device is confronted with the fact that the sound wave is transmitted thru an electrically activated metal diaphragm and is more or less distorted. I know of one gentleman who has been an otosclerotic for years and has used various types of electric devices, and finally abandoned all of them, coming back to the old fashioned wire spiral, silk covered hearing tube, and he gets more satisfaction out of that than any of the amplified radio machines.

Another factor about the electric device is, that at a distance of more than a few feet, the amplification of the voice is not satisfactory. You must be pretty close to the amplifier in order to get thoro satisfaction in the reception of sound.

I am very glad that Dr. Newhart has emphasized as perhaps the most important aid to hearing the use of lip reading. Lip reading has come into its own as a universal substitute for the adult with defective hearing. About the child, of course, there is more to be said. The adult who loses his hearing after he has thoroly mastered automatic speech is one equation; the child who has never been taught to hear and never has had an opportunity to hear requires not only lip reading as a means of contact from you to him, but he requires in addition the ability to talk, which is his contact with you. But when an adult should use lip reading should be settled by the otologist, and every otologist should be in position to advise his hard of hearing patients to master lip reading as about the only satisfactory and permanent aid to his handicap.

I appeal to you all—this is a large body, that can do a great deal of good in the propagation of a thing that is thoroly satisfactory and reliable—I appeal to you all to regard lip reading as an asset to the deafened man, a thing to have in your minds every time you advise a deafened person that you can do no more by surgery or therapy.

Dr. Robert Sonnenschein, Chicago, Ill.: This excellent presentation, concise and complete, leaves very little for discussion. I would like merely to emphasize three or four points.

With reference to mechanical aids, one should remember that Dr. Paul Sabine, of the Rivgrbank Laboratory, showed some years ago, that with the exception of the largest, (hence, most cumbersome and inconvenient) types of tubes or horns, the hand placed against the edge of the auricle, with the concavity forwards, acts just as well as a collector of sound waves and as a resonator. Years ago, I happened to obtain a few tubes made of copper, which were very light, easily carried and gave tremendous aid to hearing. They were of English manufacture, rather expensive and difficult to get, but they were the best type of hearing tubes I have ever seen.

As regards the electric hearing devices, we all realize several defects, or disadvantages. There is the rather high price, some costing $65.00, the difficulty with batteries, the great weight of some of the appliances, and the adventitious sounds produced by so many of them. These forms of apparatus give best results in middle ear, or better said, conduction apparatus impairment. C. von Eicken (Berlin), at the meeting of the German Otolaryngological Society, June, 1927, presented his findings with reference to the types of impaired hearing favorably or otherwise influenced by the electric devices. He states that those most helped were cases of advanced middle ear impairment, less those cases of combined middle and inner ear affection, and the least benefit was derived in cases of auditory nerve impairment. With this statement, I am quite well in agreement.

The matter of choice of a particular type of apparatus (provided any is advisable), is very important. Some patients respond better to one kind of hearing device (whether it be mechanical or electric) than others. It is therefore necessary, in my opinion, that the patient be permitted by the dealers not only to test out the appliance at the shop, where they may be excited or disturbed by noises, the presence of other persons, etc., and can thus not make a good choice, or perhaps even decide whether the apparatus in question aids them at all, but that they should be al-

lowed to take the apparatus home on approval for several days or weeks in order to be able to definitely determine the value of the instrument to them. I am glad Dr. Newhart finds the dealers now more willing to do this, for my experience, thus far, has been rather disagreeable. After telling the patients to buy no apparatus unless they were allowed to try it out at home, the answer usually has been that the dealer refuses such a privilege. Perhaps the publicity given this matter by the Committee, Dr. Newhart, McFarlan and Walker, has caused at least some of the dealers to see the "light."

Lastly, I wish to endorse and emphasize what the essayist has said about lip reading. Acquisition of this art proves a wonderful aid to the hard of hearing, not merely in their ability to mix with their fellows in business, profession or society, but it relieves them of the constant exacting and exhausting efforts of straining in order to hear the spoken word.

The trio of, first, a careful examination of hearing in order to make an accurate diagnosis, to pronounce a prognosis, and decide which type of hearing devise may help; second, selecting such apparatus; and third, having the individual, as soon as possible and as intensively as he can, study lip reading—this trio of endeavor will mitigate in very considerable measure the distressing disabilities of the unfortunate hard of hearing.

Dr. Sam E. Roberts, Kansas City, Mo.: There is no question about the efficiency of artificial aids to hearing. The chief difficulty we have experienced is getting the patient to wear the apparatus after it has been provided, due to a false pride. They do not want to wear this sign of a physical defect. I have assisted many patients to select a device, demonstrated its usefulness by tests beyond question of doubt and seemingly to the patient's satisfaction only to find after a few weeks that they have discontinued its use.

Dr. Newhart stated that patients should be introduced to artificial aids for hearing first. Perhaps he made that statement because his subject was artificial aids to hearing. Personally, I think they should be first introduced to lip reading. Many persons not over thirty or forty acquire lip reading readily, and become more proficient the longer they use it. In selecting the apparatus, it should be fitted to the patient's needs, and not the patient's needs to any kind of device.

Dr. John F. Barnhill, Indianapolis, Ind.: This subject has heretofore received far too little attention. However, as one notes that a large percentage of members in attendance have remained to hear Doctor Newhart's paper, it becomes evident that there is a rapidly growing interest among otologists in the management of the hard of hearing.

What that is worth while may be done for these unfortunates? Some of them may be improved by treating the ears, some may have the progress of their deafness arrested, and many are not improved at all by treatment. All those who may be benefitted in any way by physical treatment should be advised to receive such measures so long as noticeable benefit follows. There are also many among the class who are not benefitted who should, for psychologic reasons, receive occasional treatment, for to abandon them as hopeless often does great harm in leading them to take up with quackery methods and promises of the worst order. For the otologist to see such cases occasionally affords an opportunity of noting the progress of the affection and of providing some aid to hearing

at the proper time and of the most suitable type. Merely to send the hard of hearing individual to a store, with the suggestion that he buy some specified aid to hearing, is not adequate. The otologist should not only prescribe the type of aid that is most indicated, but he should also see the individual afterward sufficiently often to note the effect and to adjust the instrument to the best advantage. Not infrequently, the patient refuses to wear a device because insufficient and wearisome, when if he is encouraged and the aid properly adjusted, the result is highly satisfying. Best results are often not obtained from electric devices should the patient try to wear the aid constantly, for the reason that all sounds and all voices are conducted in a new, strange, and often tiresome way, the patient becoming excessively fatigued mentally, and greatly disturbed physically. A few hours' use of the device each day, at first, the time gradually increased as better tolerance is established, is far more satisfactory.

One benefit from a good hearing device is that the patient retains a normal voice from its correct, continued use. It is a matter of common observation, that those who hear spoken words badly very soon speak those words incorrectly and strangely. Any device that enables the patient to hear the spoken word and the quality of the speaker's voice, will cause him to retain his own voice at or near its normal.

All modern experience and observation has shown the great value of lip reading. Many cities have thoroly organized classes for teaching lip reading to the hard of hearing, which are doing surprisingly good work under the national guidance of experienced teachers like Doctor Newhart. All cities should have such classes, both in the public schools, and privately, to care for adults past school age. It is the duty of otologists everywhere to take active interest in these associations, and to encourage their promotion in cities and towns not already provided, for it is certain that proper help for the hard of hearing will come at its best only when otologists, who know more about the defects of hearing than others, do all that is in them to help these unfortunate people, who too often struggle in vain to help themselves.

DR. L. W. JESSAMAN, Framingham, Mass.: I do not think there is any particular disagreement among us as to the value of lip reading and mechanical devices, but there are some other things that have not been touched upon that this society, being the largest organization of specialists in the country, could do. That is, do some misisonary work, especially with the idea of helping the children—do missionary work among school authorities and also among the people in general, to get them to understand the value of lip reading, how much it will mean to the child who is partially deaf and has to sit in the front seat in the schoolroom in order to hear. That child is tremendously handicapped, not only in school, but also outside in everyday associations. If these children have been taught lip reading, they can pass as practically normal individuals as to their hearing. We as individuals could carry something of this sort in our minds, not only thinking of the adult, but of the smaller child. We also need teachers of lip reading, not only enough to supply the great cities, but also the smaller ones, so when we tell parents that a child should be taught lip reading, it can have the instruction without having to be sent too far away from home. Those of you who live in

the large cities, perhaps, will not have so much of that, because now in many places they have well organized classes, taught by men from larger centers; but in the smaller places, where you cannot find a teacher of lip reading, it is hard to get anyone to come into the locality to give instruction. In other words, it is largely a lack of organization. Let us keep that in mind as we go about in our daily practice, and not only try to handle our individual cases, but also try to get school authorities to do something to educate the public.

DR. HORACE NEWHART, Minneapolis, Minn. (closing): I wish to thank those who have so generously taken part in the discussion and helped to put over this subject, which to me looms large.

I would like to call your attention to the fact, that in nearly all of our larger cities, we have an organization for the hard of hearing known as lip reading clubs, or speech reading clubs. These organizations are ready to take up work with the hard of hearing where the physician's work, in the past, left off. These people are in a class by themselves, they are gloomy and depressed and discouraged almost to melancholia. They are economically in a bad way, and these organizations make it their business in life to help in the restoration of these people to normalcy. In other words, they look after their rehabilitation. I regret to say, that many of our otologists do not realize how much help these organizations can give them, and how these organizations welcome the cooperation of the otologists and their interest, as instanced by more of these deafened people being referred to these organizations for the aid which they can give. If you could see just a few of these cases I have, coming in down and out, and in a few weeks thru contact with others regaining their self confidence and being able to take their part in life, I am sure you would be surprised and gratified at the results.

PLASTIC SURGERY OF THE NOSE—THE PATIENT'S VIEWPOINT.

VILRAY P. BLAIR, M.D.

ST. LOUIS, MO.

First, I am going to apologize for this title by explaining what it means. I want to present a surgical reaction to a psychologic condition as the best answer we know, not necessarily a good one.

These pictures will likely bring up the thought that in many of the cases there is not sufficient reason for operation, but I am attacking the problem from the patient's point of view.

This is a selected group, mostly of earnest people. None were encouraged to submit to operation, and in most instances we hesitated considerably before acquiescing in their demands. The irresponsible and flippant types were eliminated wherever possible, and we have refused operation to more than we have operated upon. There are many people, and you have all seen them, I am sure, who from some real or fancied facial disfigurement are obsessed with the idea, that in their contact with others, attention is focused on some facial feature and not on their personality or what they are trying to put out to their fellows, and this obsession, which frequently refers to the nose, can be an absolute handicap. That is my reason for taking this up.

It is dangerous to approach these with any idea that their correction is a trivial matter. They are much more difficult to deal with than the making of a whole new nose, because in one case you are dealing chiefly with physical facts, in the other you are dealing largely with psychologic conditions, and in a few of them you will fail no matter what care you take.

The types of noses which we deal with are congenital, developmental, or acquired. In these days of the introduction of syphilis into parlor conversation, saddle nose is looked upon with suspicion by the observer and resentment by the victim. The retracted type gives the victim a feeling of insignificance.

There are a number of other kinds, and when you classify all that you know about, still other types will turn up.

In each instance the condition should be carefully studied anatomically, and the plan of correction made as nearly as possible to conform to the facts found. I am absolutely convinced, that in most of the cases, the patient will not be disappointed after a good operation, and in most instances where the surgeon is unable to satisfy the patient, it is because of lack of artistry in the surgery. This conclusion is based upon the observation of my own and others' results. In the last analysis, the answer, if surgical, must be pleasing surgery.

I shall not attempt to present the details of technic for it is not technic that I am trying to emphasize.

Saddle Nose. It may be the whole dorsal line that is retracted, it may be the upper part, or it may be the lower part. In these cases, it is simply a matter of sliding in some solid matter to bring the dorsal line forward. The original type of nose and the type of the face must both be considered in making the restoration, and the outline of the filler should merge into the surrounding planes and lines without any disturbing contours. The retroussé nose does not fit all faces, and there are many cases in which raising the bridge without adjusting the tip will not satisfy. In some the dorsal line should be lengthened, in others shortened, as well as raised. In all the front as well as the lateral aspect should be considered. It is the former that the patient most frequently views in the mirror, but the latter is the more evident to the friends. The introduction of the filler to the tip demands particular attention.

Lateral Displacements. These fall largely under the following headings. The fairly recent fracture that requires simply to be replaced and fixed in position; the adult or adolescent fracture which has been allowed to become fixed in the malposition, which requires freeing with a chisel; and the early childhood, usually at the time unnoticeable, fractures, that are followed by growth distortions and which are the most complicated of all to correct. You all know the history of that type of nose. The child is hurt when it is very small; the mother brings it to the doctor and asks if the nose is broken. The doctor can tell little more about it than the mother, and unless he is on his guard, he may commit himself unfortunately. Several years afterwards, it is noticed that the nose starts to turn to one side, and there seems to be almost no limit to the pro-

gressive distortion. These deformities seem to result from the septum pushing the bone and other cartilages out of shape. In each, the septum must be taken care of either before the time of the correction of the external deformity or afterwards. In some, the external deformity can be best attacked by chiseling the bony nose free subcutaneously from the other face bones, with or without freeing the septum from the maxilla. The mobilized nose is then fixed in a new position or relation either by a splint attached to the teeth or by guy wires passing thru the soft tissues to be attached to the teeth, the teeth being the only available anchorage points, and the nose is held up in the desired position until union takes place. Some of these growth deformities will demand adjustment of the lateral cartilages and the rearrangement of osteoplastic flaps.

The Retracted Nose. Of this class there are two general types; that in which the retraction is due to smallness or a shrunken condition of the soft parts, and that in which there is a retraction of the bony framework about the anterior nares. There may be a combination of both of these conditions. It may be congenital, developmental, or acquired, and certain of the first class, the shrunken nose, are definitely due to lues. It is surgically not practical to advance the maxilla by bone cutting, and in but few is it advisable to attempt to advance the septum and nasal bones, but it is quite practical to bring forward the cheeks, lips, columella and skin of the dorsum, and after mobilization, fixing them in this advanced position by sutures. In most instances the bridge can be advantageously brought farther forward with a filler, either at the time of the first operation or subsequently. In some the lining of the nose will have to be lengthened in order to permit sufficient advancement, while in others the external covering will also have to be pieced out.

If in early operation for cleft lip and palate the jaws are drawn together forcefully, there will likely result, in a certain number of cases, a lack of forward protrusion of the maxilla. If the ala is not put in proper relationship to the base of the columella, a flat nostril on that side will result. Following an early backward replacement of the premaxilla in the correction of a complete double cleft, the upper lip will be flat and the nose snubbed. In these congenital cleft postoperative deformities, the lip on one or both sides, and one side of or the whole columella can be advanced to correct the retracted lip and flattened or snubbed nose.

The essentially or relatively small nose cannot be acceptably enlarged by simply inserting a cartilage in the dorsum, tho at times we are induced to do it in the attempt to satisfy a patient whose time and finances are limited, as it is the only thing that can be done in one step in a condition that would require three or more steps to make a really worth while change.

When the tip is found to be drawn up and the nostrils everted by an external burn scar, the condition can be helped by an ellipse of full thickness of skin let in thru a transverse incision across the nose somewhere below the bony bridge.

The nose with an elongated tip can, in some cases, be treated by building out the dorsum, but usually it is preferable to shorten the lateral cartilages and septum. A symmetric droop of the tip of the nose is often associated with a dorsal convexity. The droop of the tip becomes more evident, and an undesirable breadth of the dorsum may result from the simple removal of the "hump". Under these circumstances, the dorsum can be made narrow by chiseling the nasal bones free; but to raise the tip, triplicate wedges of cartilage and mucosa, united by their bases, are removed from the septum and the lower lateral cartilages, the borders of these defects being immediately united by sutures. The unilateral droop that may be associated with poor repair of single harelip, is best corrected by removal of a crescent of skin and lower lateral cartilage.

It may be that the nose blamed by its host is not at fault; it being free from intrinsic fault, has obliquity cast upon it by some other disproportionate feature. Not infrequently it is the chin or upper lip, or a depression over the frontal area that is at fault. Sometimes both the nose and a neighboring feature share in the blame, and one cannot be advantageously changed without also dealing with the fellow culprit.

The bifid nose varies from two more or less complete noses with increased breadth between the eyes, to a slight spreading with notching in the midline. In early infancy, the double nose can be successfully dealt with, but while the ocular hypertelorism can be made less conspicuous by narrowing the bridge, it cannot be successfully changed.

Tumors, rhinophyma, etc., must be dealt with according to the condition.

The results must not only be immediately pleasing, but must be permanent to be fair to the patient. When needed we do not hesitate to make external incisions, as they are usually fol-

lowed by inconspicuous scars, but we avoid them wherever possible.

It is an interesting fact that the majority of these patients are brought to us by their parents or come in of their own volition, for the purpose of establishing or increasing their earning power.

DISCUSSION.

Dr. Ferris Smith, Grand Rapids, Mich.: As usual, Dr. Blair has presented a very imposing group of cases, in the main excellently handled, and leaves very little for one to discuss. However, there are some things to be said about the title of his paper, that is, the patient's viewpoint in many of these instances.

There are two distinct types of cases under consideration—those with minor cosmetic defects, which produce, possibly, neurasthenia or psychoses, or depression of the patient, and which frequently are much relieved by simple surgery or by more involved surgery. Occasionally one finds the patient is even more distressed after such surgery. I think in the experience of every man doing any kind of surgery can be found very distressing examples of this kind.

A youngster came to me a couple of years ago with a long, thin, pointed nose and asked for surgery, and after being refused for two or three weeks, finally, largely for the sake of getting rid of him, the nose was shortened three-sixteenths of an inch. It produced marked improvement in his appearance. He returned in three or four weeks and was insistent on having his nose restored to its original condition. After a few days, he returned with an Army 44, believing it was more persuasive than his conversation, and it was. He was finally induced to put down his gun, and we talked in a very friendly way. He told this story—that in his family was an adopted girl whom he wished to marry. She evidently did not care to marry him, and said she would not marry a man with a nose like his. After he had his nose fixed, he pressed his suit again, and she made fun of him because he had false pride. That accounted for the second return. The only way that man could be handled to keep him out of an institution was to enlist the services of the girl. The surgeon could not do anything further for him. So we talked to the girl, and the surgeon pretended to operate again, making a skin incision, and having the girl enthuse over the nose, and we sent him home satisfied for a period of months. His subsequent history was confinement in an institution. That was an extreme case, but there are cases like that, and we must be careful in choosing the cosmetic surgery for that type of case.

The next group comprises those people who are going to be improved mentally, and therefore their economic status much improved. A considerable number of people belong in that group.

There is another consideration which is of much interest to this group of men, and that is the functional improvement obtained by this surgery. Very excellent cosmetic results are obtained, and many of these

cases are improved in function, particularly those cases that have a deformed septum.

Inasmuch as Dr. Blair has not discussed technic, I can only again commend the very large and imposing group of excellent results which he showed here today.

Dr. Gordon B. New, Rochester, Minn.: Dr. Blair has done more than anyone else in this country in teaching how to do good plastic surgery.

One of the best things he has done is his method of building a nose from a forehead flap in a short time. Previously, this type of nose has always been a difficult one to take care of. I am presenting lantern slides of a patient to illustrate this method of treatment.

The patient had an epithelioma of the nose, which was removed by means of diathermy, with the resultant deformity as shown in Fig. 1. Temporarily, an artificial nose has been made for her and fastened on to a spectacle frame. The patient soon became dissatisfied with this artificial appliance and returned for plastic reconstruction of the nose.

Following the method of Neilson, modified by Blair, a flap was outlined on the forehead with definite measurements, and a graft placed in the middle portion of it that would form the bridge of the nose. The distal portion was turned on itself, following Neilson's method, and the flap brought down to the face. The area on the forehead was grafted with a full thickness skin graft. Fig. 2 shows the same patient after completion of the reconstruction following the Neilson-Blair method.

As you can see, Dr. Blair works out these difficult problems first, and then is only too willing to have us see his work, so that we may utilize his methods in taking care of these difficult cases.

Dr. T. E. Carmody, Denver, Colo.: I only wish to emphasize two or three points.

In regard to the case Dr. Smith spoke of, I had a case of the same kind only recently, and I am glad to say that I was able to leave home, and the patient has been operated since I left. I am glad he will not return to me when I get back, especially if he should happen to carry a 44, because I might be inveigled into doing something that my conscience would not approve.

I think Dr. Blair's way of expressing what these people look like is very good, especially when they look like totem poles and things of that kind. I think that is original with him.

Cases that want to be restored to the original condition probably give us more trouble than any other kind. If we can please ourselves with a case, I feel the patient should be pleased, because I believe we are the hardest to please. Nevertheless they are frequently displeased when the nose is almost perfect, and many times are pleased with a nose that is far from perfect.

I was especially gratified with Dr. Smith's classification, speaking of the different cases that want cosmetic restoration, and those that need restoration from the standpoint of function. I think as rhinologists we see a great many cases that are not deformed externally and in which we improve the function, and when we find a case that is deformed externally and we also improve the function, the patient should be pleased.

It is gratifying to see the results Dr. Blair has obtained in cases of total destruction. It is much better than anything we have been able to obtain, and the case Dr. New showed is absolutely perfect. I only wish I knew how as well as Dr. Blair.

Dr. Samuel Iglauer, Cincinnati, O.: The psychology of these patients is very interesting. I now have a patient that I operated on before I left home. She wrote me she was one of three sisters and was the "ugly duckling," and therefore had an inferior complex.

Another girl came out from New York. She sent her photograph in advance, and after she arrived, she was followed by her younger sister, who said she wanted her nose fixed also. I refused to operate on the younger girl who came with a governess. The latter then thanked me, and told me that this girl always wanted, and usually got, everything her sister had, and if the sister was to have a new nose, she wanted one too.

The late Dr. Loeb took me to task for operating these cases for psychologic reasons. He was of the opinion that this was not the proper way to treat them. Nevertheless, such patients are often benefitted by an operation. One was a stock broker who became more successful after the operation, but I do not know whether the benefit came from the operation or from a rise in the market.

Actors constitute a class of people upon whom we must operate. They now want to get into the movies, and often they do not photograph well, and if we improve their appearance, they have a better chance. The point has been brought out several times, that the patient is often pleased if we are not. Of course I make it a rule, if the patient is satisfied, not to say much about whether I like it or not, but very often what pleases the patient does not please me.

Another thing that has increased the amount of this work is the automobile. A great many people have their faces disfigured in automobile accidents, and we must learn how to take care of them. There will be a lot of this work to do, and it is up to us to do it.

Dr. Earl C. Padgett, Kansas City, Mo.: A Chicago surgeon of the highest standing a short time ago stated to me that he had just returned from France, Germany and England and had taken particular interest in finding out what was being done in plastic surgery. In his opinion, the work being done in St. Louis was superior to the work being done elsewhere.

The psychologic aspect of deformities is extremely important, and that of facial blemishes is most important of all. Rather rapid strides have been made in our knowledge of the socalled "mental complexes" generally. Altho probably not to be completely accepted, the behaviorists have added considerably to our knowledge of the end results of conditioning an individual to unusual and unnatural environmental stimuli. Surgeons very often have little or no knowledge of these various conditioning influences and the final results as far as the attitude of the patient to his environment is concerned. Undoubtedly, in the approach to treatment, we should borrow some of the knowledge of the psychiatrist and the psychologist.

Dr. New mentioned the technic of building a complete nose. Altho the forehead flap offers the easiest method, I am not at all sure it is the best. I have one patient with a good nose built from a forehead flap.

I have another patient with a forehead too narrow to get a good forehead flap. Therefore, a flap was raised from her arm, but the type of tissue found on the arm was much more difficult to control than the forehead flap. The nose was not a great success, and I cut it off. On the second attempt, I built the complete nose on the arm before throwing it up on the face. The lining was placed in by turning a second flap backward in under the flap forming the covering of the external nose. Rather early, a cartilage was transplanted from the chest into the flap. Due to the stiffening the cartilage gave the tissue of the flap, it was more easily controlled. This gave a good shaped nose. The disadvantage of a scar on the forehead was thus obviated, but the color of the nose was somewhat whiter than the surrounding face tissue.

Dr. Joseph C. Beck, Chicago, Ill.: I have had a good many disagreeable experiences in cases where I had to do this work. I have not turned all of them away, but I have kept quite a few away (you may laugh, but it is true) by telling them that I would pattern their nose after mine.

I have discussed this matter with Professor Neymann (professor of psychiatry at Northwestern University) seeking information as to whether these cases were psychic, and if so, whether the psychic case could be treated any other way aside from operation. (Dr. Neymann and I are preparing an extensive article on this subject.) I long ago adopted the method of making casts, both plaster and wax, keeping the plaster cast for record and employing the wax model to make the correction. I always make the correction less attractive in the neurotic individuals, hoping to drive them away, but it does very little good. Dr. Blair's idea is undoubtedly the right one. What they like, is what we should give them, reoperating if necessary. I feel that the work Dr. Blair is doing is individual. I am sometimes tempted to do work like this, but I feel that I should quit it and leave it to a man such as Dr. Blair, who is competent and able to handle it. One of the discussers referred to someone who said that his (Blair's) work was ahead of anything in the country. It might have been I who said it, for I have made such a statement a good many times.

As to the question of the quacks, including doctors who have fallen by the wayside and gone into quackery (we have them in Chicago), I think if an ethical expert man like Dr. Blair does this work, there would be less of the unfortunate conditions develope that cause people to fall into the hands of the quacks.

Someone spoke of actors and professional people. I recall a case I recently had, a young lady whose nose and lip I changed by operation, and she is today on a circuit in vaudeville because she has a wonderful voice. I was pleased and so was she, because she was able to secure this good position, which prior to the operation she was unable to secure. I could not use cartilage or bone in that case, because it was a nose with a pathologic membraneous lining and I was afraid of infection, consequently I used ivory, and it is staying all right.

The question of artificial noses was mentioned and the statement was made that people would not wear them. If a case cannot be operated and still there is great deformity, as in healed out carcinoma or after syphilis, you can use a material that has been developed in Germany and is now

being used quite extensively by Dr. Lederer in Chicago. It is a material not unlike rubber, and the patient makes his own nose as often as required. They are made from dies. They are colored a little corresponding to the rest of the face, and people wear these where they cannot have an operation. I would like to have Dr. Blair's view on that procedure, and this time would like to pay tribute to Dr. Blair and to the work he has done.

Dr. Vilray P. Blair, St. Louis, Mo. (closing): That certainly was a handpicked bunch of discussion. But it gives me a good feeling when I hear it.

Dr. Beck spoke of the nose made of a gelatin and glycerin preparation, fastened to the face with a screw, so that the facial muscles in moving will give movement to the nose, and if the patient will follow the directions and take the trouble to take care of it, it gives surprisingly good results. I happened to be in a position in the army where I could keep track of the ultimate outcome by tabulation of the majority of these cases, a great many in the British Army. The Government spent quite a good deal of money getting ready to make this artificial appliance, and it seemed to be a success over there, but I do not know a single case in the American Army where they have been willing to go on wearing an artificial appliance. I do not believe that many of them will take the trouble to keep up an artificial nose. It has struck me, that those people who are willing to wear an artificial appliance soon forget about it entirely, and will go on wearing it with the paint worn off and all dented in—wearing it like a child wearing its mother's old dress—perfectly happy.

The matter of psychology has been mentioned. It is rather encouraging, the acknowledgment that one is not doing his full duty by simply telling these people to forget it. This type of surgery has been hurt a great deal by another type of psychology, and that is to do something to the nose or any part of the face which is not a successful piece of work, and then try to put it over and induce the patient to believe that it is. That is a mistake. It is remarkable how far in times past that has been put over.

THE RELATION OF SINUS DISEASE TO HAY FEVER.

T. E. Carmody, M.D. and L. W. Greene, M.D.

DENVER, COLORADO.

The literature available on this phase of the subject is very limited, and it appears that this most important condition has been very lightly treated; then, merely considered as unimportant, with the exception of Schadle, who was our pioneer in 1907, and some few men who have recognized the possibility that infected membranes in the various sinuses might be a contributive factor in hay fever. We take the stand, that without infection in the sinuses or without pathologic membranes, our irritants, such as dusts, pollens, bacteria, foods and emanations of all sorts, etc., do not produce the socalled allergic coryza or hay fever.

We began our work on the subject by making the pollen tests in all cases. We used the pollen extract treatment with varying results, but in no case did we effect permanent relief. In many cases, the condition was aggravated. One of us, at that time, complained of the classical symptoms of socalled "hay fever." Shrinkage of the nasal membranes alone did not give relief. A simple puncture of one of the maxillary sinuses, with irrigation, did give relief, however, from the symptoms which had been complained of for years before, altho flakes only were recovered. One of us complained of these same symptoms, having been tested and found sensitive to the common ragweed. Simple puncture and washing of the maxillary sinuses relieved this condition. This gave us the incentive to find out if we could not relieve others so afflicted.

Our attention was drawn to a method which might prove more efficacious, i. e. we began a series by testing and X-raying all of our cases which showed clinical signs of "hay fever." The pictures proved in our first few cases that the membranes were involved in varying degrees.

In our large series of cases, the patients presented themselves with the classical symptoms and history of exposure to the various irritants. Some had complained of these eymptoms recurring each year, for periods varying from one to twenty years. These,

in most instances, of course, were seasonal and due to the various pollens. In our locality, the most common irritants are the ragweed group, the thistle group and the sage brush group. The clinical history in all cases gave the information, that during the winter months they were comparatively free from symptoms. Schadle, of St. Paul, in 1907 recognized the causative factor and attributed the infections of the antrum as the cause. Fortunately we were able to start our work by skin tests. In this series, due to the interest of the late Dr. Dean Beacom, Associate Professor of Clinical Diagnosis, University of Colorado, these cases were tested by him, then referred to us for clinical and X-ray check-up. In no case did the clinical and X-ray findings disagree, which is proof enough that pathology exists.

The next step was to see if these cases could be relieved. Treatment consisted of the usual shrinkage of the nasal membranes, etc. Many responded to the icthyol treatment, others to simple puncture of the maxillary sinuses, and others (five in number) to radical operations. This work has been observed for the past three years without failure of results in any case.

We are all familiar with the various theories concerning "hay faver," namely: the toxin theory, the protein sensitization theory, and the hereditary theory. Our concept deals with "Membranes and Filterable Causative Agents," which may or may not be a combination of the above theories or entirely independent.

The entire body, as we well know, is covered by a continuous protective enveloping membrane. This envelope, when not suffering from pathologic change or insult due to violence, is more or less impervious to outside influence. This phase of the subject does not take in the influence of toxins manufactured in the body itself and brought to the eliminating organs.

Death certainly, and possibly life itself, seem to be measurable by the parity of balance maintained between the factors which determine the nonentrance of outside elements, except food, oxygen, and factors which assure perfection of elimination. A skin that is what we term "alive" is "water proof," but the nearer it approaches a condition of dissolution, the more fluid or infection permeable it becomes. The paper thin intestine stands year after year as an almost miraculous barrier against infection, until some pathologic departure from normal renders it filterable to a toxic intestinal content.

The conjunctiva, altho exposed thru life to outside irritation and infection, remains while normal and while bathed in that re-

markable antiseptic, the tears, an impregnable defense; but let the continued absence of vitamins rob the conjunctiva of the elements necessary for its maintenance, and it becomes an easily broken barrier with small protective properties. A nasal mucous membrane which is normal, is (in consequence) resistant and nonfilterable to outside protein invasion, but it becomes after greater or less period of abuse, due to faulty metabolism of protein from focal infection or faulty tissue chemistry, a spongy hypertrophied dropsical tissue, threaded with dilated capillaries, which give us our symptoms of socalled "hay fever." This membrane is now potentially a pathologic tissue and readily permeable to pollen proteins.

Most pathologic hypertrophies have back of them the very item which accounts for physiologic and natural increase in size and added amount of work. In a pathologic hypertrophy, the added work is called for because the tissue is compelled to do unnecessary and abnormal duty in the metabolism of faulty chemical substances brought to it.

If we visualize the one great item of capillary caliber and physiologic blood vessel elasticity as a determiner of normal function, and classify a great majority of failures in anabolism and catabolism due to protein influence as "urticarias" of the tissue involved, many pathologic states will be rendered more easily understandable. A skin urticaria, a "big tonsil" or a large boggy turbinate, adenoids or generalized edematous nasal mucous membrane which allows a weed pollen protein to filter thru it, a chronic or acute laryngitis, a bronchitis or asthma, are all departures from normal due to protein anaphylaxis, and all show the unstable dilated capillary. This may be accounted for by the biochemist with the acid combinations which compel engorgement of membranes, etc. Whether from sinus or other point of focal infection, such as food ingested, the results will practically be the same in any susceptible individual, namely, "local tissue dropsy."

The cleaning up of a faulty body chemistry which has compelled an "urticarial" condition in given tissue, necessarily protects from further protein invasion thru these tissues. In "hay fever," it would seem logical to ignore the weed pollen protein producing the distress, and instead, to look to previous infection of, and keep the sinus mucous membranes in a state approaching normal and throw up a natural and physiologic barrier which cannot be broken down by extraneous invasion and "bombard-

ment." No normal mucous membrane is filterable to an outside protein. If in given cases, the emptying of an infected sinus and its subsequent healing brings about clinical cessation of socalled "hay fever" symptoms because the nasal tissues are no longer required to metabolize pus proteins which render them abnormal, would it not be a logical procedure to believe we are dealing with pathology of these membranes before an irritant comes along?

Treatment of these cases is divided into three heads: those relieved by simple dehydration of nasal mucous membranes by the use of a 20 per cent solution of ichthyol and glycerin tampons; those relieved by simple puncture of the maxillary sinuses; those relieved by radical operation on the sinuses. In our series, seventy-seven responded to the simple ichthyol tampon treatment, nineteen to the simple puncture of one or the other or both maxillary sinuses, and five to the radical operation on the maxillary sinuses and exenteration of ethmoids.

In summarizing, let us recall that we have realt with but one phase of this vast subject, namely, "hay fever" as regards sinus infection. And we have found that the maxillary sinuses, as Schadle pointed out twenty years ago, are the most commonly infected. Next in frequency are the ethmoids, frontal sinuses and sphenoid sinuses.

Slide No. 1. Rogers—12—0-1—Treatment about every ten days.

Slide No. 2. Carrigan—21—H. F. for 5 yrs. before. Treatment only. (Max. 1-2.)

Slide No. 3. Sayre—25—3-4—Double radical; bad membrane.

Slide No. 4. Boae—18—Max. 3-1. Double radical (2-4-28).

Slide No. 5. Wagner—27—Incomp. oper. before; reop. by us, O. K.

Slide No. 6. Heverley—22—rt. Entire membrane detached. Seven yrs. H. F.

Slide No. 7.

These are just a few of our many cases to prove our theory but our time will not allow further detail.

DISCUSSION.

Dr. John J. Shea, Memphis, Tenn.: During hay fever, the mucous membrane of the sinuses is in a state of vasomotor relaxation and it reabsorbs its own discharge. The irrigation of the antra with the best continuous drainage possible will reduce this absorption. We have used autogenous vaccine made during the acute stage for years, with better results than the use of pollen antigen started after the onset of an attack.

Many of the hay fever victims have chronic sinuses, and a study as to whether the sinuses were first diseased or whether the vasomotor condition produced it is a worthy one.

Dr. T. E. Carmody, Denver, Colo.: There are only two or three points that need to be emphasized here. The first one Dr. Greene touched upon at the end of the paper that this is only one phase of the subject. We might go into asthma and other pathologic conditions, but we do not wish to do so at this time.

Another point is the number of cases that are relieved by the use of pollen extract. Only 40 per cent are relieved according to the statistics of men who use pollen, using it before the seasonal attack.

Dr. Shea mentioned that this was simply an allied condition. We believe the disease in the sinuses or nasal mucous membrane is the primary cause, and the protein from the pollen dust or whatever irritant is instilled is simply the exciting cause—that we have the pathologic membrane first. However, we may possibly have the reverse. But in our experiments we have found that a great many of these sinuses in children are affected early in life, long before the child has hay fever, and in all of these cases we found evidence, both clinically and by Xray, of disease of the sinuses. We have found a number of sinuses infected within the first year of life, and in one case we found the sinus infected in a baby eight days old—not a hay fever case, but it simply laid the foundation for other pathology.

The only thing we have to consider, is what is the cause and what is the effect? It happened that one of us received great relief after washing the sinuses, being relieved entirely for a week, even tho he went into the weeds with the pollen that caused the acute attack. That was evidence to us that the infection in the sinus had a great deal to do with it.

TRANSACTIONS

OF THE

THIRTY-THIRD ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

PRESENTATION OF INSTRUMENTS AND APPLIANCES

THE ZEHENDER-GREEN SINGLE CELL TRIAL FRAME.

A. E. Ewing, M. D.

ST. LOUIS, MO.

The employment of plano lenses in the practical correction of refraction was first suggested by Professor Karl W. Zehender[1] in 1866. In 1878, Dr. John Green[2] devised a thin flanged rim for the trial lens, in order that lenses of low power as well as plano lenses might be placed face to face in a single celled trial frame. The suggestions of these pioneers in refraction for this arrangement of lenses in the trial frame was a long step forward in the adjustment of lenses to the eye, as has recently been proved in the Committee work of the American Ophthalmological Society under the direction of Dr. Wilmer, "On the Ophthalmic Powers of Lenses," in which Dr. Shahan demonstrated the varying values of the claims of opticians for their numerous special lenses. It was shown in the work of this Committee, that so far as concerns their practical value to the patient, negligible variations existed in the best of these lenses, but the demonstration went further to show that slight variations in tipping the trial lens, or in moving it nearer to or further from the eye, caused appreciable changes in the focus of the lens, which could prove detrimental in the adjustment of the lens.

Unquestionably, our above mentioned pioneers in refraction appreciated these fine differences in lens adjustment from actual experience in their office work, and, searching for the cause and discovering it in the underlying principles of refraction, they were prompted to suggest methods of arrangement of lenses so that their axes and focal points would be more in unison with the focusing mechanism of the eye. The value of such arrangement is self evident to the ophthalmologist or to the optician who desires that the lenses in his trial case may be placed before the eyes of the patient in essentially the same combination as will finally be prescribed.

This end is easily accomplished by means of plano lenses fitted with thin flanged rims and fixed in a light trial frame. The

lenses may be worn comfortably for a short time, until the accommodation becomes quiet and the eyes have adjusted themselves to the new condition. Thru such trials, the ophthalmologist may assure himself that his work will be satisfactory.

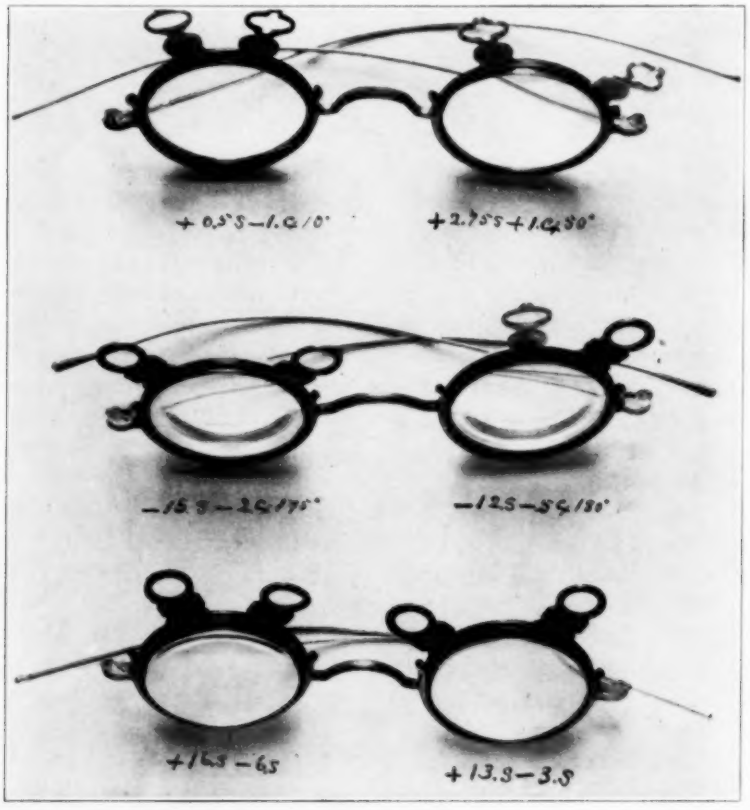

Fig. 1. Illustrates the Zehender-Green trial frame and shows combinations that may be made with it when the trial case is supplied with Zehender plano lenses mounted in the thin flanged rim devised by Dr. Green.

In honor of the eminent men who first suggested these improvements in trial lenses and in the mounting of trial lenses, I have christened this frame the Zehender-Green light trial frame.

The first example of it was made for me by Mr. A. P. Erker of the Erker Bros. Optical Company, by enlarging the groove in the ordinary single cell steel frame by the process of grinding. This method was found to be very difficult because of the hardness of the tempered steel. Mr. Erker then prevailed on the

American Optical Company to manufacture a lighter and a better frame than any on the market. A number of these frames were obtained, and with their aid greater accuracy has been achieved in all compound astigmatic corrections, than is possible with ordinary lenses.

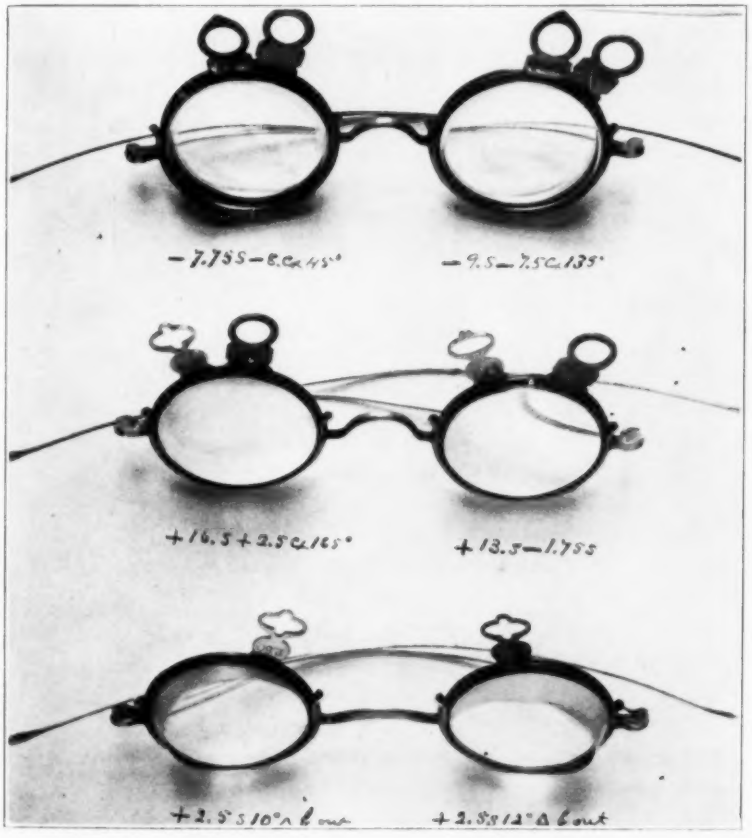

Fig. 2. Shows further combinations with the Zehender plano lenses mounted in the Green thin flanged rim.

As an example, a patient has been under my care for several years who has a compound astigmatism of nine diopters in each eye, the actual correction being O. D. + 0.5 spr. — 9. cyl. axis 44° V = 20/24 +, O. S. + 2.25 sph. — 9. cyl. axis 126° V = 20/20 +. This very high grade of astigmatism was readily corrected by means of the trial frame and plano lenses, and the correction is entirely comfortable to the patient.

In all cases of aphakia, and in all cases of very high grade myopia or hypermetropia complicated in any way with astigmatism, plano lenses and this trial frame are especially valuable. Also, in any case in which a meniscus lens is desired, the plano convex lens may be placed in the front portion of the groove and may then be backed by a plano concave lens of any curvature desired. (This concave lens is usually spherical, — 3. or — 6.)

The essentials for such a frame are elasticity and lightness in weight in the cells, and the temple should have sufficient springiness to hold the frame comfortably on the face. Also, there should be numerous forms and widths of bridges to fit all kinds of noses and all pupillary widths.

Because of its strength, lightness in weight, and elasticity, steel is the best material out of which to construct the frame, but it has the fault of corroding very readily, at least in St. Louis atmosphere. Also, it is acted on by the perspiration of the face. To obviate this tendency to corrode, I have tried plating with nickel, silver, and gold, and lately with chromium. Nickel, silver, and gold quickly wear thru. Moreover, most of these metals contain alloys which are acted on by the perspiration of the face, so that they leave a stain on the bridge of the patient's nose if the frame is worn for an hour or so. The chromium has been tried for only a short time, but thus far it has proved to be more satisfactory than the other metals.

Altho Zehender suggested the employment of the plano lens in refraction so many years ago, and a half century ago Green[8] further demonstrated its practical worth by mounting plano lenses with thin flanged rims, this valuable means of doing the best possible work in refraction does not seem to have come into general use, the reason being, I am told by opticians, that plano lenses are somewhat more expensive. This expense is, however, only about $50.00 to $80.00 more on a standard trial case that would cost $160.00. I am further informed by one of our largest manufacturers of optical appliances, that about one plano lens is sold to five hundred double convex and double concave lenses for equipment of trial cases. This is a sad reflection on intelligent refraction. It is an evidence of the low standard for refraction demanded by ophthalmology. Long experience with both forms of lenses has taught me that the difference in price is a bagatelle in comparison with the time saved, alone, by employing plano lenses, and if ophthalmology generally were aware of how much more accurate such lenses are when used in the manner indicated

by our great men in refraction, who really understood refraction in all its fine variations, the ratio in the trial cases would soon be reversed, and refraction in this country would be greatly improved.

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1. Zehender. Klinische Monatsblaetter f. Augenheilkunde, vol. IV, p. 7.
2. Green. Transactions Am. Ophthalmological Society, vol. II, pp. 483-488.
3. Green. Transactions American Ophthalmological Society, 1880, 16th Meeting, p. 133.

CHROMIUM PLATED LANCE POINTED MAGNET TIPS.

A. E. Ewing, M. D.

ST. LOUIS, MO.

In the majority of injuries to the eye resulting from bits of magnetic metal, a small opening is made by the metal as it passes into the globe. For the removal of such foreign bodies, the magnet is usually employed, under the influence of which the foreign body is drawn toward the opening and may pass into it and be extracted. As a rule, however, these foreign bodies are more or less ragged and angular, and they are very liable to hang in the

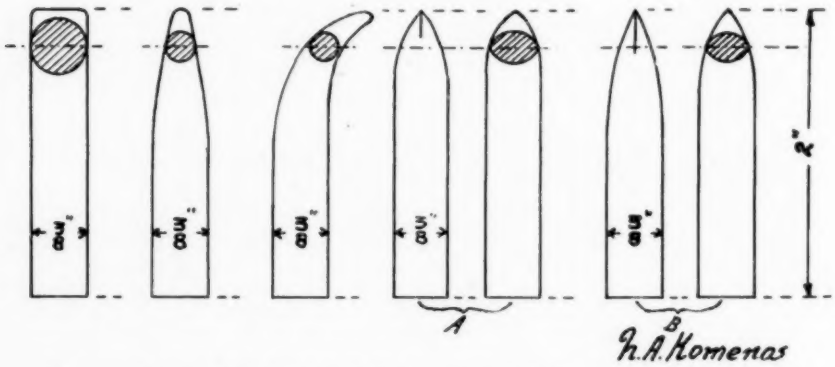


Fig. 1. Lance pointed magnet tips in contrast with tips ordinarily employed. At A and B the sides and edges of the two forms of the lance pointed tips are illustrated.

tissues. This is a serious hindrance to the use of the magnet, because any tissue between the tip of the magnet and the foreign body will produce a resistance that is not easily overcome even by strong magnets. Also the movement of the foreign body in the tissues causes more or less injury and often pain. For these reasons, it is advisable to construct the magnet tip so that it may come immediately in contact with the foreign body with the first introduction of the tip into the wound. In order to accomplish this with facility, I have for many years employed a lance pointed tip which will enter a small opening and at the same time contain a mass of iron sufficiently large not to interfere with the efficiency

of the magnet. Properly sterilized such a tip may be passed into a corneal or a scleral wound without harm or danger to the eye, and if the foreign body is floating in the vitreous or is anywhere near the wound, it will make immediate contact with the tip and be withdrawn.

Should the foreign body hang in the wound, as it frequently does, this form of point has another advantage, in that it may be turned in the wound, thus making a larger opening for the exit of the foreign body.

For the construction of tips for the magnet, the best metal is soft iron, which, however has an objectionable feature in that it corrodes very readily, and when the tips are suddenly wanted, they are found to be covered with rust. To overcome this defect I have tried plating the tips with silver and with nickel. The nickel peels, and the silver turns black. Recently I have tried chromium, which is proving to be very satisfactory. It does not peel and is not easily stained with either mercury bichlorid or sodium chlorid solutions.

CALIBRATED STEREORADIOGRAPHIC UNIT.

W. P. Reaves, M.D.

GREENSBORO, N. C.

Last year, at the Detroit meeting, I exhibited a Shadowgraphscope, Marker and Calibrated Angle Head Table.

When the table was used with a not calibrated tube stand, the angles and position could only be registered approximately.

To register the technic of stereoradiographs requires a Calibrated Stereoradiographic Unit of Tube Stand and Table combined.

In the short time that I have today, it is my pleasure to show you a Calibrated Stereoradiographic Unit, which is a Tube Stand and Table combined, which completes the instruments necessary for the technician to register on the film his technic of positions and angles and other data, so that the observer can optically duplicate the positions and photographic angles in the shadowgraphscope shown you last year. It also enables the observer to study by comparison in the transparent image the true topography (eliminating distortion), third dimension and details of abnormal anatomy or pathology, even tho he has not been clinically trained to interpret shadows of abnormal anatomy or pathology.

When the Head Specialist says to the Roentgenologist "Let me see instead of show me," it may be explained because he is trained in optics, orientation and head pathology.

The illustrations in last year's Transactions and this will enable one to check up the merits of X-ray technic from target to transparent image.

Note: The positions in Fig. 1 are maintained.

Fig. 2 shows that the target can be made to rotate around the head and cassette tunnel. The same principle is now applied and supplied to the cassette tunnel, which enables one to make exposures with patient on his back, cassette on right side and target in position shown. For exposures of the left side both cassette and target are reversed.

These two universal adjustments are adaptable for any position of the head desired. As in Fig. 1; Fig. 2 lying on the back, or in the natural sitting position, the head vertical, as preferred by many.

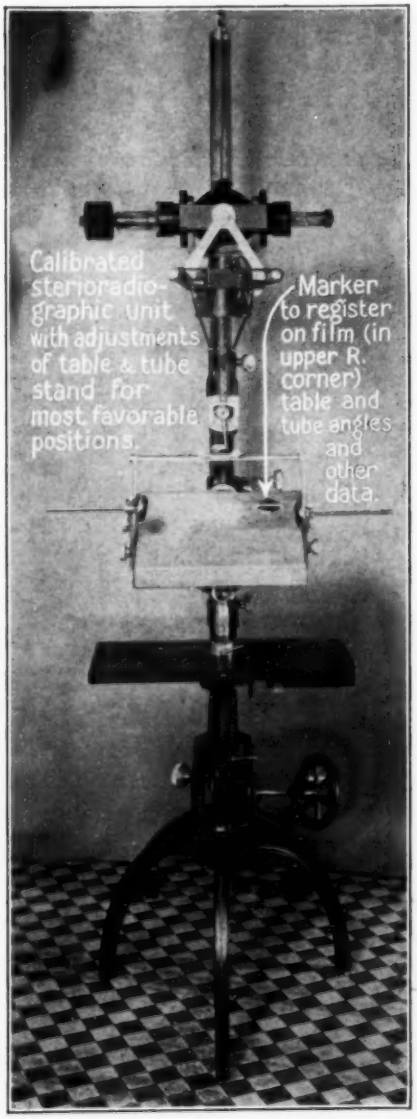

Calibrated sterioradiographic unit with adjustments of table & tube stand for most favorable positions.
Marker to register on film (in upper R. corner) table and tube angles and other data.

Fig. 1.

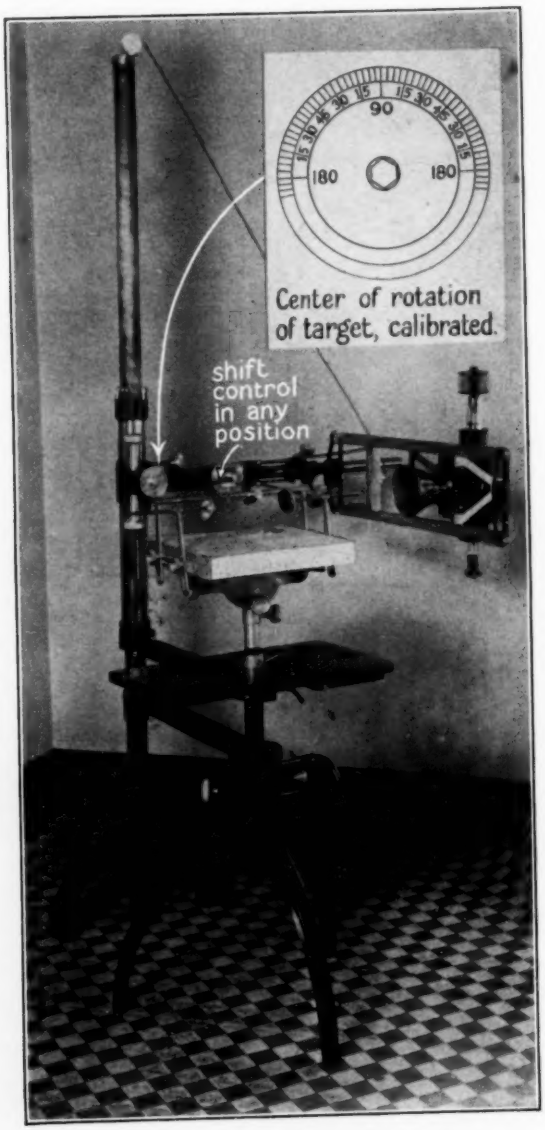

90
180
180
Center of rotation
of target, calibrated.
shift
control
in any
position

Fig. 2.

CYLINDRICAL HANDLES FOR INSTRUMENTS FOR CATARACT OPERATIONS.

DANIEL B. KIRBY, M. D.

NEW YORK CITY, N. Y.

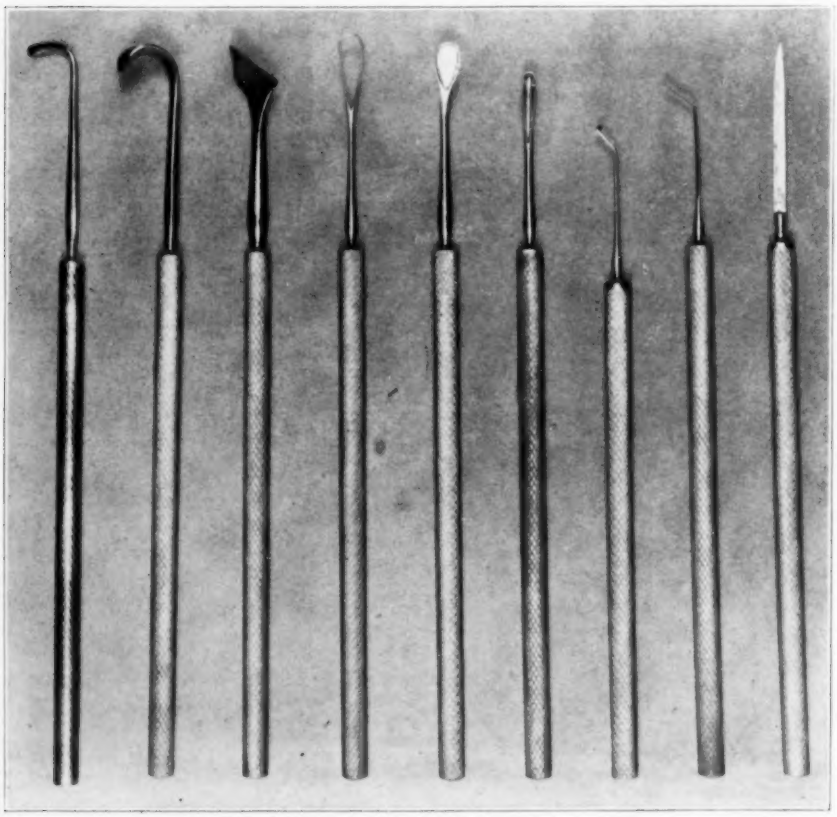
In studying out the technic of the use of the iris repositor, it was found that the flattened or squared handles did not lend themselves to an easy adjustment for all positions. It was thought that a cylindrical handle would make possible the finest adjustments, being controlled with the thumb, forefinger and second finger. A 5 mm. diamond knurl round or cylindrical handle was devised by the author for the iris repositor. It proved to be of value for the technic desired. While the flat or square handle often required an entire readjustment of the hand to suit the instrument, the round or cylindrical handle was easily adjusted to suit the hand in its most natural position. Undoubtedly, cylindrical or round handles have been used before in cataract surgery. This handle was chosen by the author as the most satisfactory one for him.

MATCHED OR RELATED INSTRUMENTS FOR CATARACT OPERATIONS.

DANIEL B. KIRBY, M. D.

NEW YORK CITY, N. Y.

It seemed that it would be a great advantage if the same handle were had for all the instruments used in any operation requiring skill and delicacy of movement. If every instrument had precisely the same feel, the same grip, length, weight, size and



Instruments are shown ¾ of natural size.

balance, it would not be necessary to make repeated adjustments, because of differences in these factors, in instruments successively used in a cataract operation.

Accordingly, the Graefe knife, keratome, cystotome, lens spoon and loop, lid retractor, hook and iris repositor were designed with matched or related diamond knurl round or cylindrical handles.

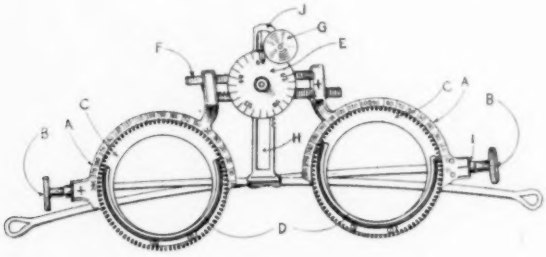
Dr. John M. Wheeler designed a cylindrical handle for the cystitome which he devised. It was while studying under his direction, that the author devised these new instruments. The matched or related instruments have been very satisfactory in practice. They were made according to the author's design by the E. B. Meyrowitz Company.

THUMB SCREW TRIAL FRAME.

S. B. MUNCASTER, M. D.

WASHINGTON, D. C.

While studying in Vienna, in 1889, with my friends, Drs. Wilder, Lancaster, and Wuerdemann, it occurred to me that it would be a great help if we had a trial frame in which we could have a spherical lens, and at the same time have a cylindrical lens that could be rotated by means of a thumb screw, and thus avoid having one's hand in front of the patient's line of vision when attempting to find the cylindrical axis. I talked this over with Dr. Wuerdemann and asked him to help me design the frame, and he contributed the movable nose rest and pupillary distance



attachment. The result of our efforts is shown in the accompanying diagram and photostat of the frame. A is a stationary cylindrical ring upon which the cylindrical markings are placed; attached to its posterior surface is a spring (as D), with its open portion down so as to hold in a spherical lens. Over the anterior surface is a separate cylinder, C, upon which the cylindrical lens rests and is rotated in either direction by a cog wheel system controlled by the thumb screw, B. Upon this cylinder is a spring, D, which holds the cylinder in place as it is rotated on C. The temples, I, are attached to the posterior surface of A as is shown in the diagram. The rest of this trial frame was designed by Dr. H. V. Wuerdemann.

The nose rest moves in the vertical plane and is adjusted by means of the thumb screw, E, and the pupillary distance is read directly from the thumb screw by means of the marker, J.

The trial frame I have described is the first one made, I believe, upon which the axis of the cylinder could be controlled by means of a thumb screw. My mechanic wished to get a patent but I would not permit this, for I preferred to give it to the medical profession without the stigma of a patent by a physician.

However, in 1895, Henry H. Hempler of Washington, D. C., secured a patent for a sight adjuster, without furnishing a model, similar to the frame I designed six years previous.

I believe my model of a trial frame was the forerunner of the present day trial frames with their many modifications and improvements.

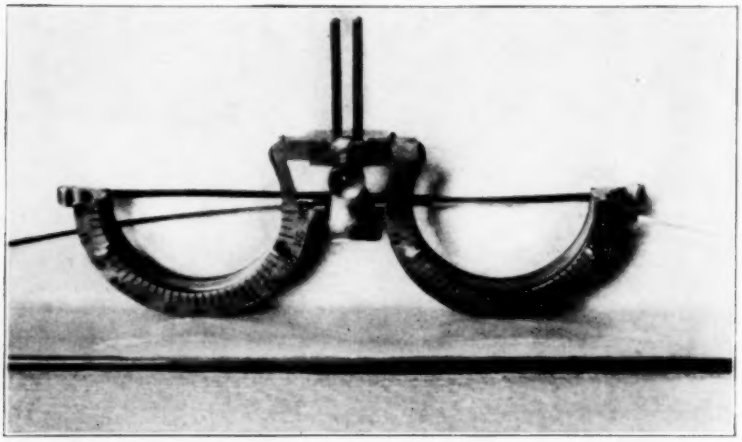
(Mr. Mueller, of Chicago, says this frame (made about 1889) is hand made and could not now be duplicated for less than $200.00. Its original cost was about $15.00.)

TRIAL FRAME.

WILLIAM M. BANE, M. D.

DENVER, COLO.

I desire to bring before you a trial frame designed by my father, Dr. William C. Bane, of Denver. It is something like the trial frame designed by Ivan Fox some forty years ago, which



father has been using with a good deal of satisfaction all these years. This one, we believe, is somewhat of an improvement on the Fox frame. Its advantage is that it is very rigid, is easily adjusted, is accurate for corrective lenses, and will take a cylinder 1½ inches in diameter. It is made to hold three pairs of lenses. It is manufactured by the Denver Experimental Works.

FORCEPS FOR BARRAQUER-GREEN CATARACT OPERATION.

OTIS WOLFE, M. D.

MARSHALLTOWN, IOWA.

The instrument is a combination of the Fox and Green forcep. It is convenient for those who do the Green modification of the Barraquer cataract operation. We employ the Green instruments and technic.

When the corneal section is picked up to insert the erisphake, there is no danger of it slipping away, as with a lighter forcep.

The conjunctival flap can be held firm when the sutures are inserted so there will be no pull. (Described in former paper.)

If the section should come out short of the limbus, the cornea can be firmly grasped and held while Kalt needles and sutures are inserted thru the slot. It is heavy enough to handle the needle if so desired.

After the erisphake attaches itself to the lens capsule, the forcep is rotated in the fingers and the ball tip used to make counter pressure at the limbus as described by Green.

The instrument is held in the left hand and used for all the steps of the operation. The operator need not distract his attention by making a change of instruments.

NONMAGNETIC FOREIGN BODY INSTRUMENT.

NELSON M. BLACK, M. D.

MILWAUKEE, WIS.

The instruments I have to present are nonmagnetic, for use in removing foreign bodies from the eye. The one in particular that I wish to call to your attention is a bayonet shaped knife. When a foreign body is located or brought into the anterior chamber, and an incision made for its removal, the aqueous humor is lost and the foreign body either disappears, becomes

entangled with the iris, or moves to some other portion of the anterior chamber and it is difficult to again bring it to the point of incision. If a linear incision is made, the probabilities are that it will be impossible to extract the foreign body without enlarging the incision. The idea of this nonmagnetic instrument is that it makes a "leech bite" incision. The magnet can be kept in contact with the eye at point of incision, and as the aqueous escapes, the traction of the magnet will pull the foreign body out of the anterior chamber.

TONSILLAR PILLARS RETRACTOR.

W. D. Black, M. D.

ST. LOUIS, MO.

I have designed an instrument for retracting the tonsillar pillars, to be used after the tonsils have been removed. In my hands, I find the hemorrhage is controlled to a great extent, and it is much easier to catch the bleeding vessels when necessary.

After spreading the pillars, one can see the floor of the tonsil fossa thoroly. You can use it on either side—on right side downward and left side upward. The handle is adjustable.

The instrument is very crude at present, but Mr. Mueller of the V. Mueller Instrument Company, is going to make a much better looking instrument, but with the same mechanism.

ADENOID FORCEPS AND KNIFE.

JOSEPH C. BECK, M. D.

CHICAGO, ILL.

I am presenting an instrument, as yet unfinished, for the removal of adenoids. This is the first time that I have presented an instrument in an unperfected state. However, it will not be manufactured or placed in production until it has been thoroly tried out. I intend to observe its efficacy for adenoidectomy for at least a year, and in addition, gather reports from several large

clinics where it will be used and compared with other popular adenotomes.

It is a combination of an adenoid forcep with a knife arrangement that cuts the adenoid tissue off. The forcep is opened by a spring, and the adenoid grasped by the jaws of the instrument which is then closed. By proper manipulation of the knife, the adenoid tissue is cut from above down. The instrument is submitted at this time merely to be placed on record. Mr. Wallerich, who has aided in the technical work on the instrument, will demonstrate it in the commercial exhibit.

TRANSACTIONS

OF THE

THIRTY-THIRD ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

NECROLOGY
MINUTES
DIRECTORY
INDEX

DEATHS

Dr. Joseph Zepher Bergeron, Chicago, Ill.
Dr. Edward Leonard Bull, Ithaca, N. Y.
Dr. Walter Almond Hager, South Bend, Ind.
Dr. Edward Henry Higbee, Jr., St. Louis, Mo.
Dr. Frederick William Lamb, Cincinnati, Ohio
Dr. Henry Hager Martin, Savannah, Ga.
Dr. Frank Athon Morrison, Indianapolis, Ind.
Dr. Richard H. Satterlee, Buffalo, N. Y.
Dr. Greenfield Shader, St. Louis, Mo.
Dr. Herbert G. Tureman, Kansas City, Mo.
Dr. Leon Edward White, Boston, Mass.

MINUTES.

October 16, 1928.

The thirty-third Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology was held at the Hotel Statler, St. Louis, Missouri, October 15th to 19th, 1928.

The Tuesday morning session was called to order at nine o'clock by the President, Dr. Luther C. Peter of Philadelphia.

THE PRESIDENT: Ladies and Gentlemen: It gives me great pleasure to call to order the thirty-third meeting of the American Academy of Ophthalmology and Oto-Laryngology.

I have one unfortunate announcement that I know will distress you all, namely, that our Guest of Honor, altho not ill, is not able to appear on the program this morning. He feels, however, that he will be ready to present his paper tomorrow evening at the Business Meeting. I am sure we all hope that Dr. Jackson will recover promptly and be able to be present at that time.

I wish to announce, that at the close of this morning's session, Dr. Harrison, of Philadelphia, will present a very interesting series of moving pictures of ophthalmic operations. I am sure you will all be interested in seeing these pictures.

The first item this morning is announcements by Dr. H. W. Lyman, Chairman of the Local Committee.

DR. H. W. LYMAN: The first announcement I wish to make is that the St. Louis University School of Medicine invited all the members of the Academy to be its guests at luncheon today at twelve-thirty. The medical school is three and one-half miles from this hotel, and after a careful consideration by the officers of the Academy of the difficulty of transporting the membership to the school, and the impossibility of getting them back in time for the afternoon session, the luncheon has, with regret, been called off. When the situation was explained to Father Schwitalla of the University, he concurred in the wisdom of this procedure.

I want to call attention to the banquet, which takes place in this room tonight at eight o'clock, and to request that you buy your tickets for this banquet as you leave this room this morning.

Tomorrow night there will be the business meeting in this

room, and to this the ladies are also invited. As soon as the routine business is out of the way, we have arranged for the showing of a prereleased picture. This picture has not been publicly exhibited, and we are told it is very fine.

Thursday night will be given over to special dinners, and anyone wishing to arrange for such special dinner should get in touch with the management of the hotel. You will be well taken care of.

At a banquet given at the Chase Hotel Thursday evening, the Dana medal for the prevention of blindness will be presented. The members of the Academy are cordially invited to attend.

The Wintersteiner exhibit of 10,000 sections of pathologic eyes will be shown at the St. Louis University School of Medicine. Visitors will be welcome, and there will be an attendant to aid those desiring to examine the collection.

Following the announcements, the President read his Address, and a symposium was presented on recent developments in neurology as it applies to the eye, ear, nose and throat. (See Table of Contents, Joint Sessions.)

DR. GEORGE F. SUKER, Chicago: I move a rising vote of thanks to our guests for the privilege we have had of hearing their discussions. Motion seconded and carried.

THE PRESIDENT: The Chairman would like to add his personal appreciation of the courtesy of these men in helping us out. The Tuesday morning session then adjourned.

BUSINESS SESSION.

October 17, 1928.

The annual Business Meeting of the American Academy of Ophthalmology and Oto-Laryngology convened in the Ball Room of the Hotel Statler, St. Louis, Missouri, at eight o'clock, Wednesday evening, October 17th, 1928, Dr. Luther C. Peter, President of the Academy, presiding.

Following the announcement of the President's luncheon, Thursday noon, the following reports were read:

REPORT OF EXECUTIVE SECRETARY.

The work in the Secretary's office during the past year has proceeded without friction. Drs. Benedict, Myers, and Gradle have each assumed entire control over the work of their respec-

tive sections—the Executive Secretary has not been called upon to interfere. All of the executive officers, in doing their work, have kept the Secretary informed thru copies of letters, with the end result that a maximum of work has been done without confusion or conflict.

I want personally to express my appreciation to all of the executive officers for the splendid cooperation manifested. Especially do I want to thank Dr. Peter for the way he has worked, has thought out problems, and has cooperated with his secretaries.

At the Detroit session the membership was listed as	1,403
Elected, 1927	80
Total	1,483

List of Candidates, 1927, contained 120 names, audited as follows:

79 became members at Detroit.
1 later admitted (Grant).
3 refunds—Huntz, Thorpe, von Zelinski.
23 old applicants carried to 1928.
14 old applicants written for renewals.

120

Those carried forward (23 plus 14—37) are accounted for in the following way:

6 certificated.
24 carried forward—not certificated.
6 dropped (applications over 3 years old).
1 withdrawn (Townsend).

37

During this year we have instituted a loose leaf Membership Ledger which will serve as a permanent record, containing date of admission to the Academy, specialty practiced, financial relations, and other data.

There are now carried on the books:

Members in good standing		1393
Honorary members		12
Life members		11
One year delinquent members		36
Two year delinquent members		5
To be dropped for nonpayment of dues		4
Carried by order of Council		5
Making an actual membership of		1466
Deceased during the year	11	
Resigned during the year	8	
Dropped during the year	3	22
		1488
Applicants pending:		
Complete	133	
Incomplete	37	170
Total carried on books		1658

DURING THE LAST TWELVE MONTHS.

Death has claimed eleven Fellows, as follows:

Dr. J. Z. Bergeron.
Dr. Edward L. Bull.
Dr. Walter A. Hager.
Dr. Edward H. Higbee, Jr.
Dr. Frederick William Lamb.
Dr. Henry H. Martin.
Dr. Frank A. Morrison.
Dr. Richard H. Satterlee.
Dr. Greenfield Sluder.
Dr. Herbert G. Tureman.
Dr. Edward Leon White.

The following eight Fellows resigned:

Dr. C. B. Bliss.
Dr. W. H. Dudley.
Dr. A. E. Hubbard.
Dr. J. W. Kimberlin.
Dr. Seymour Oppenheimer.
Dr. Haskell S. Phelps.

Dr. P. N. K. Schwenk.
Dr. Hiram Woods.

Three have been dropped for nonpayment of dues:

Dr. Roy R. Norton.
Dr. H. L. Stelle.
Dr. D. C. Louchery.

The number of applicants carried this year is the largest in any one year of the Academy's existence.

In accordance with the By-Laws, the dues are divided: five dollars credited to operating account, and five dollars to research account.

There passed thru the Executive Secretary's office during the fiscal year just closed:

Operating receipts		$15,486.86
Operating costs		16,345.80
Showing an operating deficit of		858.94
Research accounts receipts. (This does not include interest on our capital investments handled by the Treasurer)		$ 7,200.00
Less research appropriations	2,000.00	
Less operating deficit	858.94	
	$2,858.94	
Net passed to capital investment		$ 4,341.06

At the Detroit session, the Executive Secretary was instructed to advise the Crerar Library, Chicago, that the Academy was willing to enter into negotiations with them, to the end that Academy members could have the use of their facilities.

Two letters were written and a personal interview was had without any particular interest being manifested on their part.

Respectfully submitted,

WILLIAM P. WHERRY,
Executive Secretary.

There being no objection, this Report was received and filed.

REPORT OF THE TREASURER.

August 6, 1928.

The American Academy of Ophthalmology and Oto-Laryngology,
Cleveland, Ohio.

Dear Sirs:

In accordance with instructions received, we have examined the accounts of your Academy for the period from August 1, 1927, to August 1, 1928, and submit herewith a summary of receipts and disbursements for the period together with a statement of the assets at August 1, 1928. We also attach a schedule of bonds owned at the latter date. The $5,000.00 par value Union Square Company, First Mortgage Leasehold 6½ per cent bonds owned by the Academy are in default, consequently they have not been included in the list of assets attached hereto, altho the loss to the Academy will probably not amount to the full par value thereof.

All receipts and disbursements were traced to the Academy's bank account. The balance in the bank at August 1, 1928, was verified by a certificate from the bankers, and the bonds on hand August 1, 1928, were counted by us.

The following is a summary of the changes in the financial status of the Academy during the past year:

Assets held August 1, 1927		$77,991.53
Excess of Receipts on Income Account over Expenses:		
Receipts	$22,572.39	
Expenses	16,187.56	6,384.83
		$84,376.36
Union Square Company bonds written off		5,000.00
Assets, August 1, 1928, per attached statement		$79,376.36

Yours very truly,

Price, Waterhouse and Co.

SUMMARY OF RECEIPTS AND DISBURSEMENTS FROM AUGUST 1, 1927 TO AUGUST 1, 1928.

Cash in bank and on hand, August 1, 1927		$ 4,020.03
Receipts:		
On Income Account:		
Dues from members and applications for membership	$14,900.00	
Income from exhibitors	3,445.00	
Interest on bonds, bank balances, etc.	4,025.39	
Profit on bonds sold	202.00	
	$22,572.39	
On Capital Account:		
Bonds sold—cost	7,073.00	
Total Receipts		$29,645.39
		$33,665.42
Disbursements:		
On Expense Account:		
Office Expense	$ 4,712.55	
Expense of meetings	8,334.14	
Research work	2,961.70	
Accrued interest on bonds purchased	179.17	
	$16,187.56	
On Capital Accounts:		
Bonds purchased	12,972.25	
Total Disbursements		29,159,81
Cash in bank August 1, 1928		$ 4,505.61

SUMMARY OF ASSETS, AUGUST 1, 1928.

Cash in bank	$ 4,505.61
Bonds owned, per attached list	74,870.75
	$79,376.36

SCHEDULE OF BONDS OWNED AUGUST 1, 1928.

Description	Interest Rate	Due	Par Value	Cost
Kansas City, Fort Scott and Memphis Railway Refunding Mortgage ..	4 %	—1936	$ 5,000.00	$ 4,352.50
Chicago, Rock Island and Pacific Railway First and Refunding Gold Bonds	4 %	—1934	6,000.00	5,337.00
Midvale Steel and Ordnance Co. Convertible Sinking Fund	5 %	—1936	15,000.00	14,707.50
Swift and Company Sinking Fund Gold Notes	5 %	—1932	3,000.00	2,907.00
American Telephone and Telegraph Company Sinking Fund Gold Notes	5½%	—1943	4,000.00	4,026.00
Denver Gas and Electric Co. General Mortgage..	5 %	—1949	4,000.00	3,951.00
Witherbee Sherman Company First Mortgage Sinking Fund, Series "A"	6 %	—1944	5,000.00	4,925.00
Van Sweringen Company First Mortgage and Collateral Trust Notes ...	7 %	—1930	9,000.00	8,910.00
Southern California Edison Co. General and Refunding Mortgage ..	5½%	—1944	4,000.00	3,865.00
Commonwealth Edison Co. First Mortgage and Collateral, Series "C"..	4½%	—1956	5,000.00	4,825.00
Detroit City Gas Company First Mortgage Gold Bonds, Series "B"	5 %	—1950	5,000.00	5,025.00
Kingdom of Denmark Gold Bonds	5½%	—1955	4,000.00	4,040.00

Youngstown Sheet and Tube Company First Mortgage	5	%—1978	3,000.00	3,002.25
Columbia Gas and Electric Co. Debenture Bonds..	5	%—1952	5,000.00	4,997.50
			$77,000.00	$74,870.75

There being no objection, this report was received and filed.

REPORT OF THE EDITOR OF THE TRANSACTIONS.

Seventeen hundred copies of the 1927 Transactions were printed at a total cost of $3,744.97. From this should be subtracted $23.00 for reprints furnished gratis to the guest of honor, $103.50 for reprints of our List of Members, and $193.28 received from various authors for their half share of the cost of the cuts for illustrations, leaving a balance of $3,425.19, or an average of $2.01 per copy.

The fact that the meeting was held in the middle of September and that the Transactions were in the mails by Christmas, was due to the promptness with which most of the speakers returned their discussions or corrected proofs. A few, however (chiefly chronic offenders), did not return their matter until the Transactions were being printed, so the Editor was compelled to use his own revisions in order not to delay its appearance. Since the meeting is a month later this year, it will be impossible to get the Transactions out by the end of the year, *unless* the speakers return their discussions and reports *within the ten-day limit*.

The printer has in storage the following back numbers of the Transactions: 1912, 10; 1916, 13; 1917-18, 5; 1919, 78; 1920, 88; 1921, 102; 1923, 95; 1924, 652; 1925, 171; 1926, 111; 1927, 100.

Any of these may be obtained from the Editor upon prepayment of $5.00 per volume (nonmembers, $10.00). The Editor suggests that an attempt should be made to obtain a complete set of the Transactions to be kept either in the archives of the Academy, or in the John Crerar Library, or in the Library of the Surgeon-General.

This year the Academy issued, for the first time, a Pre-session Volume, consisting of the papers to be read before the Oto-Laryngologic and the Ophthalmologic Sections. The first papers were received by the Editor August 13th, and the last

one August 26th. Some papers were never received, and consequently had to be omitted. Most of the corrected proof was received from the various authors well within the time limit, but one was not received until September 19th. As this particular paper happened to be one of the earlier ones in the book, it was impossible to page the majority of the papers until it had arrived. Consequently, the books could not be mailed before October 2nd. The Editor submits that from August 13th to October 1st, one and a half months, is too short a time to allow for all the contingencies that might arise in the preparation of a Presession Volume. At least a month must be allowed for editing and typesetting, depending upon the number and length of papers. At least another month is necessary to send proof to the authors, receive their corrections, compare with the Editor's, revise and page, print, bind and mail. It can easily be seen what possibilities exist for fracture of a schedule.

If the Academy decides to continue the publication of a Presession Volume, the copy should be in the hands of the Editor at least two and one-half months before the date of the meeting to *insure* the copies being in the hands of the members two weeks before the meeting. This means that the author should have his paper and illustrations in the hands of his Section Secretary three months before the date of the meeting, and should return his corrected proof promptly.

The Editor desires to explain, that the amount of space left at end of each paper for comments, in no way represents the value of the paper, but is dependent on its length, and the desire to start each paper on a right hand page and leave at least one blank page for comments.

Seventeen hundred copies of the Presession Volume were printed. By arranging to have the type held set-up, to be used in printing the 1928 Transactions, the cost of printing and mailing will be about $1,000.00 more than the Transactions alone would cost.

Respectfully submitted,

CLARENCE LOEB,
Editor.

There being no objection, this report was received and filed.

REPORT OF THE SENIOR MEMBER OF COUNCIL.

It is my privilege to submit, for the Council, the following report of its activities:

It recommends for Life Membership, Dr. Hiram Woods of Baltimore.

It reports the receipt of a communication from Mr. Harding of Cleveland, relative to the establishment of a fund to be offered to the discoverer of a cure for glaucoma. This has been referred to the Council's Research Committee for a later report.

A motion was passed expressing to Dr. Max A. Goldstein the thanks and appreciation of the Academy for his courtesy and services in giving, in the Laryngoscope, special publicity concerning the arrangements for this meeting of the Academy.

The Secretary was instructed to express to Mrs. Sluder the condolences of the Academy in her bereavement thru the very recent death of her distinguished husband, Dr. Greenfield Sluder of St. Louis.

A motion was passed appointing Thomas Cook and Sons as the official travel bureau for the Academy in connection with the coming International Congress of Ophthalmology in Amsterdam, in 1929.

The Council voted to make available for research during the coming year the sum of $4,000.00: $2,000.00 in the field of Ophthalmology, and $2,000.0 in the field of Otolaryngology, all expenditures to be subject to the oversight of the Committee on Research. It was voted that $500.00 of this sum be appropriated as a contribution to the support of the Army Medical Museum.

It was voted to continue publication of the Presession Volume of the Transactions.

The incoming President was authorized to select the Guest of Honor for the 1929 meeting.

Approving the recommendation of the Finance Committee of a change in the handling of the finances of the Academy, the Council gives notice of proposed amendments of the By-Laws covering these points.

The Council further gives notice of a proposed amendment to the By-Laws increasing the number of active Fellows on the Council from three (3) to four (4), each to serve for a period of four years instead of three years.

It is also proposed to so change the By-Laws as to provide that when a member is elected to Fellowship in the Academy with the certificate of one of the national examining boards, he will be classified in the official membership list of the Academy as practicing the specialty for which he holds the certificate, and will not be classified as both an ophthalmologist and an oto-

laryngologist until he shall have received the certificate of both Boards.

The Council approved the recommendation of the Committee on the Hard of Hearing, that the Academy place itself on record as favoring the early passage in our several States of a uniform law which shall effectively provide for the regular yearly testing of the acuity of hearing of all school children, by such standardized methods as shall be adequate to detect even slight degrees of hearing loss, as an initial step in the prevention of deafness and the conservation of hearing.

The Secretary was instructed officially to thank the management of the Hotel Statler for the very efficient and courteous manner in which they have provided for the needs of this convention.

The meeting place for next year will be Atlantic City.

The following nominations are made:

President	Dr. Harris P. Mosher	Boston
President-elect	Dr. William H. Wilder	Chicago
First Vice-President	Dr. Frank E. Burch	St. Paul
Second Vice-President	Dr. Harry W. Lyman	St. Louis
Third Vice-President	Dr. John H. Dunnington	New York
Secretary	Dr. William P. Wherry	Omaha
Treasurer	Dr. Secord H. Large	Cleveland
Member of Council	Dr. John J. Shea	Memphis
Editor of Transactions	Dr. Clarence Loeb	Chicago
Associate Secretary Section for Ophthalmology	Dr. William L. Benedict	Rochester
Associate Secretary Section for Otolaryngology	Dr. John Myers	Kansas City
Secretary for Instruction	Dr. Harry S. Gradle	Chicago

Respectfully submitted,

HORACE NEWHART,
Senior Member of Council.

REPORT OF COMMITTEE OF FINANCIAL PLAN.

It has been well known for some time, that the scheme of carrying on the financial part of the Academy could be improved upon, altho we seem to have a fairly healthly balance in our reserve. At the Detroit meeting, a committee was appointed to

devise a financial scheme. This involved the following changes in the By-Laws, which are submitted at this time.

Article 4, the Treasurer. Strike out the entire old paragraph and substitute the following:

"The Treasurer shall approve all bills authorized by the Council and sign checks jointly with the Secretary, before same are valid. He shall keep account of all expenditures and shall present a statement to the Academy at its annual meeting. He shall be under reasonable bond to be determined by the Council."

Also under Article 4, The Secretary, add to the present By-Law the following:

"He shall collect all money due the Academy and deposit same to the Academy account."

The method of withdrawing these funds for expenses or investment is detailed in the appended financial plan.

1. Single bank account where moneys are collected.
2. All expenditures must be authorized by the Council before incurred, except that regular current expenses and small accounts originating at conventions may be paid and approved at the next Council meeting.
3. All bills to be paid by check with signatures of both Secretary and the Treasurer.
4. Monthly financial statement sent to the President, Treasurer, and members of the Council.
5. An annual audit by an expert Auditor.

INVESTMENTS.

1. The President, Treasurer, and Secretary to constitute the Investment Committee. This committee to be limited in its choice of investments of those securities which would be acceptable in trust accounts. (Note): (This would mean bonds of the Government, State and certain municipalities. Most trust companies recognize certain bonds of large corporations and occasionally a first mortgage on real estate.)

2. All securities belonging to the Academy to be held by a suitable trust company, which will collect all interest and deposit it to the Academy account. No security to be sold or exchanged without the consent of the entire Investment Committee.

SAMUEL E. ROBERTS.

THE PRESIDENT: Dr. Wheeler will clarify the situation as to the proposed amendments for next year.

DR. JOHN M. WHEELER: The Committee on Constitution and By-Laws have no further report to make.

THE PRESIDENT: The several items reported by the Senior Member of Council will be taken up in their proper order.

At this point we propose to arrest our business program and at once proceed to the scientific part of the evening session; then we will return to the business. We are happy to state that Dr. Jackson is here, as you can see, and that he will be able to present his paper.

For some years past we have followed the custom of inviting as a Guest of Honor one who is recognized as an authority in Ophthalmology or Oto-Laryngology, or a specialist in some phase of these branches of medicine. We have had many distinguished guests from abroad. Last year we digressed from our usual custom and honored one of our own members from our neighboring country, Canada. This year we feel that we have honored ourselves by inviting one of our own members who was born, reared, and educated in his profession in the United States. We feel particularly happy in our choice and in his acceptance, because no guest from at home or abroad could serve in this capacity more capably or acceptably than Doctor Edward Jackson.

His work as an ophthalmologist, as an author, and as a teacher in his special branch, is so well known, that an attempt on my part to dilate upon his accomplishments would tell you nothing which you do not already know. An introduction is manifestly unnecessary. It affords me much pleasure, however, to be able to present to you our distinguished Guest of Honor for this year, Doctor Edward Jackson, who will speak to us on one of his favorite themes—"Refraction."

DR. EDWARD JACKSON: Fellow Members of the Academy—for I shall not forget that we are fellow members: I wish to assure the many who expressed their regret that I was not able to be present last night, that no one regretted it more than I did.

I thought very seriously of a subject on which to speak—something of value that would in a way justify the unusual invitation I have received. I felt when I chose this subject, and feel still, that it is a most important one, and if I can to some extent impress the thought of its importance upon you this evening, I will feel justified not only for the work I have put upon it, but also for my absence last night.

And in addition, I want to say that no expression of esteem and good will ever touched me more than the two members

appearing at the door of my room with the immense bouquet made up of one flower taken from each table at the banquet. I do not think I was ever more touched by any such expression. (See Table of Contents Joint Sessions.)

DR. E. C. ELLETT: As a further evidence of the professional and personal esteem in which our Guest of Honor is held by the members of this society, I move that Doctor Jackson be made a Life Member of the Academy.

Motion seconded by DR. ARTHUR J. BEDELL, and unanimously carried by rising vote.

THE PRESIDENT: By this vote of the Academy, Doctor Jackson, your name is placed upon our Life Membership roll rather than upon the roll of Honorary Members. This is done with design, in order to keep you in close affiliation with all the activities of the Academy.

In addition to this expression of appreciation, the Council feel that they have properly interpreted the wish of the membership at large and have, therefore, unanimously authorized more concrete testimonial to the value of your contribution to the Academy and to Ophthalmology in general, and more concrete evidence of our devotion to you, personally, in the form of the highest award which lies within their power to give, namely, the Academy's medal. This testimonial is in the form of a paper weight, which will adorn your desk in the future.

I trust that as it holds in place the many papers and editorials which we hope may continue to flow from your facile pen, it may recall to you not only evidence of approval of your work and your contributions to our beloved science, but that it may especially convey to you the love and esteem in which you are held by each and every member of the Academy.

I have the honor, Sir, by order of the Council and of the membership, to present you this Medal of the Academy.

DR. EDWARD JACKSON: Fellow Members of the Academy: I did not believe that it was possible for the Academy to heap on me any more honors, for I feel I have been more than sufficiently honored before. I am entirely unable to reply adequately to this new manifestation. I can say truly, however, that as we grow older we do get more pleasure from the appreciation and good will of those who have known us well and remain our friends.

Report of Committee on Arrangements.

Dr. H. W. Lyman: The Committee on Arrangements have very little to say in the way of a report. We have tried to provide everything for the convenience of the sessions, and if our arrangements have enabled the meetings to be conducted successfully and have contributed to your pleasure, we are content.

Report of Program Committee.

The Program Committee met in St. Louis, Sunday, December 18th, 1927. The meeting was called to order by Dr. Peter.

Present: Drs. Peter, Dennis, Post, Lyman, Myers, Gradle, Wherry.

The question of a Presession Volume was considered. It was decided to try the experiment for the St. Louis session—this in accordance with the wishes of the Council. It was further decided to allow each essayist five minutes to present the subject matter of his paper, and several additional minutes for slides.

It was agreed not to publish the papers of the symposium in the Presession Volume.

The Program Committee recommended that Dr. Lawrence Post be appointed as Associate Editor of the Transactions, should Dr. Loeb wish an assistant.

The question of having a President's Reception on Sunday night, October 14th, was left to the decision of Dr. Peter.

After mature deliberation, it was thought advisable not to combine the conferring of the Dana Medal with the annual banquet.

The program presented at this meeting was decided upon.

Respectfully submitted,

Wm. P. Wherry,
Executive Secretary.

The Secretary: The President instructs me to announce that the Program Committee will meet in December at some central point to arrange a preliminary draft for the Atlantic City meeting. It is hoped that all who wish to present papers or participate in the next meeting of this body will communicate with the proper Section Secretary. It is rather the thought at present to send out a preliminary Fall notice to the members asking for the cooperation of the membership of the Academy

with the Program Committee in constructing the program for next year.

Report of Committee on Standardization of Tuning Forks and Hearing Tests.

To the Members of the American Academy of Ophthalmology and Oto-Laryngology:

Your Committee last year, at the meeting held in Detroit, called attention to a new type of fork which has been developed by Mr. B. E. Eisenhour, at the Riverbank Laboratory in Geneva, Illinois. We believe our society, and all otologists, owe the proprietor of this laboratory, Col. George Fabyan, and his scientific staff, a great debt for the courtesy and assistance always shown your Chairman in his efforts to get information which might be of aid to our specialty.

The Committee, in reports made a number of years ago, stated that one of the requisites of a good fork should be its construction with metal which is rust proof. When forks are unnickeled they rust, unless carefully oiled, and if nickeled, when peeling occurs, the vibration of the loose pieces of metal produces adventitious sounds. Many types of metal, including stainless steel, have been used, but these forks have usually been lacking in the best qualities, such as pureness of tone and long duration of vibration. Forks of an alloy of magnesium, which seems to fulfill most requirements, were mentioned at the time of the report in 1927, but the exact composition of this alloy was not then known to us. We are now reliably informed that the metal consists of magnesium 95.6 per cent; manganese .4 per cent, and aluminum 4 per cent. Since the specific gravity of steel is about 7.7, that of aluminum 2.7, and that of magnesium 1.7, it will be seen that this alloy is extremely light, its specific gravity being about one-third that of steel. The question of lightness is extremely important, for when one is using the large low, or medium, pitched steel forks, the fingers become very tired holding them in their proper position, especially when the Rinne test is performed and the fork is held at right angles to the mastoid process.

With reference to brittleness, we are informed that there has been no more breakage from the manipulation of these forks during the past year than is usual with the steel forks. The lowest forks have long prongs, with a tendency to produce rather pronounced and persisting overtones, but this is much less than with some of the steel forks, especially if the fork is struck at

the proper point with a soft substance, such as a cloth hammer or the side of the hand. Furthermore, overtones may be eliminated by means of weights, such as are used on the lower pitched steel forks, or merely placing about the prongs in the region of the percussion point a wide rubber band, such as Bezold originally recommended for his unweighted A (108 double vibrations) fork.

So far as the duration by air conduction is concerned, the magnesium alloy forks are practically as good as steel ones, except the c-4 (2048 d. v.) and the c-5 (4096 double vibrations), especially the latter. At some of the pitches, the magnesium forks are even better than some of the steel ones, but on the other hand, some of the medium low pitched steel forks vibrate a little longer.

So far as bone conduction is concerned, it was thought, when reported by your committee a year ago, that bone conduction with these forks was not as suitable owing to their light mass. However, some tests done by one of us (Sonnenschein) showed that for bone conduction the magnesium forks do very well; in fact, there was very little difference noted between the magnesium and the steel forks. Further tests are being made in order to settle accurately these points regarding the comparative value of the magnesium and steel forks with reference both to air and bone conduction.

Thus, a series of these forks has been perfected from 16 to 4096 double vibrations. In other words, as complete a range is covered by magnesium alloy forks as hitherto possessed by the best steel forks. So far as the price is concerned, these forks in all probability will cost less than first class steel ones, but surely not any more.

The damping "constant" can be determined for each fork at a cost of about a dollar and a half, which is somewhat lower than the charge made by the Bureau of Standards for steel forks. It was pointed out in the Committee Report of 1926, that by having this "constant" of damping, or decrement, it was possible to obtain the percentage of loss of hearing figured in sensation units, just as is done with the audiometer.

The greatest advantages of the magnesium alloy forks are those of being rust proof, and extremely light in weight. We believe that the inception of this type of fork is a most decided advance and will probably prove of great advantage to the otologist. Whether they will eventually actually replace steel

forks, we cannot at present state. In view of the importance of this subject, we believe this new type of fork should be intensively studied, so that we may be able to come to a definite conclusion regarding it.

The Committee presents this report, and recommends that the Committee be continued in order to carry out further study and investigation of these new forks, and other matters affecting the functional testing of hearing.

Respectfully submitted,

L. W. DEAN
GEO. W. MACKENZIE
E. G. GILL
ROBERT SONNENSCHEIN, CHAIRMAN.

October 17, 1928.

There being no objection, this report was received and filed.

REPORT OF OTOLARYNGOLOGIC BOARD.

DR. T. E. CARMODY: The members of the Board in this society, Dr. Wherry and myself, are part of the entire Board of ten members. Since the last meeting of this Academy, the Board has held five meetings and four examinations, and in the four years that we have been in existence, we have examined about 1600 applicants.

REPORT OF OPHTHALMIC BOARD.

DR. W. H. WILDER: The American Board of Ophthalmic Examinations, since the last meeting in Detroit in 1927, has conducted examinations at Memphis, at Minneapolis, at the time of the American Medical Association, and one in this city last Monday. The examination in Memphis was held November 14, at the time of the meeting of the Southern Medical Association, and was a very satisfactory examination. At that time, ten candidates were examined. On June 11, 1928, at St. Paul, fourteen candidates were examined, and at the meeting here last Monday, 33 candidates.

The number of certifications in 1927 was 60; in 1928, 49, making a total of 687 who have been certificated by this Board.

We are contemplating holding an examination next July at the time of the meeting of the American Medical Association in Portland. Publication of this will be made at the proper time, so as to give prospective candidates ample opportunity to complete their arrangements.

We would like to have it generally understood, and at this time make the announcement, that if at any time, in any given locality in the country, enough individuals can be found who are interested in this matter of having an examination, the Board will make provision for such special examination, if we can hold it in some large center, where ample clinical material is available, because we rely upon that for the examination, which is partly written, but more largely practical and oral.

The Academy is now represented on the Board by:

Dr. John M. Wheeler.....First term ends 1/1/1929
Dr. F. P. Calhoun.....Second term ends 1/1/1930
Dr. Jas. M. Patton.....First term ends 1/1/1931

We are glad to see, from the action of Council, that Dr. Wheeler is eligible for reelection.

I am glad to notice that the Council has deemed it wise to take under consideration the very important condition that obtains in this society, where we have specialists of two kinds—ear, nose and throat, and the eye. It seems to me, it is a very wise suggestion that is made by the Council, that members of this large organization who make profession of being specialists in all of these head specialties should be required to show their eligibility to such specialties, and hence, it is only proper that those who make such profession such receive the endorsement of both Boards, and should therefore take both examinations—or if not that, some form of general examination which might be devised for the purpose.

I wish to announce, in closing, that the Board still has on hand a number of copies of the Directory of certified ophthalmologists thruout America. It is arranged alphabetically and also geographically, so that if you wish to refer to a competent ophthalmologist in some part of the country, you can turn to the Directory. It is the same kind of Directory as put out by the Otolaryngologic Board. We shall get up before the first of the year a supplementary list, so as to bring this Directory to date, and to everyone owning such a Directory, we shall send the supplementary list gratis. This saves the additional expense of getting out a new Directory

REPORT OF COMMITTEE OF PUBLICITY AND LEGISLATION.

Your Committee on Publicity and Legislation presents the following report:

During the year we have sent to every medical journal at least one notice of this meeting. Most of the publications have printed an abstract of the session's program. We believe this to be a valuable aid in stimulating interest and securing a larger attendance of the members at our sessions.

The local committee on publicity has been of inestimable assistance to your committee. They arranged newspaper conferences, and advertised the objects of our association.

Various legislative activities have been reviewed, but we have not attempted to influence legislation.

Two things demand our consideration:

The Opticians' Guild of America have started a campaign of education and have introduced for general use the term "eye surgeon." For excellent reasons, we propose the designation "eye physician," and urge the adoption of the appellation by the Guild.

The second matter is that of medical advertising. We are cognizant of the many ramifications of this evil, we appreciate the sectional customs, and yet the appearance of advertisements stating the price of treatment is, we believe, so fraught with danger, that as a Committee we look with disfavor upon the recent innovations. Your Committee, therefore, moves that it be the sense of this Academy that it disapproves of the publication of fees in any form, whether intended for professional or lay readers.

ARTHUR J. BEDELL, CHAIRMAN.

DR. A. J. BEDELL: While I am on the floor, as the arrangements for this meeting have been so wonderfully carried out, I move a rising vote of thanks to the local Fellows and the ladies for their constant efforts in our physical, mental and moral behalf. (Motion unanimously carried.)

REPORT OF COMMITTEE ON OPHTHALMIC AND OTOLARYNGITIC PATHOLOGY.

The Section on Ophthalmic and Otolaryngitic Pathology has been functioning since 1921, in conjunction with the Army Medical Museum. During that time. we have received sections, examined, described and reported upon 918 specimens. We have received 689 eye specimens that had already been examined and diagnosed when received. We received among other items, partly thru the generosity of the St. Louis members, 1127 specimens,

making a total of 3777 specimens. These are exclusively ophthalmic. That is since 1921. Therefore we have today in the Army Medical Museum a very respectable pathologic collection of which we are proud.

	Eye Specimens Received and Reported	Eye Specimens Diagnosed When Reported	Other Items, Illustrations, Models, Etc.	Total
1921	23	...	23	23
1922	129	11	679	819
1923	83	95	73	251
1924	161	4	256	421
1925	136	191	4	331
1926	105	...	...	105
1927	163	262	89	534
1928	118	46	3	167
	918	689	1127	3777

NOTE: The number of eyes for 1928 includes the specimens of eyes received up to September 1, 1928.

You may remember that we are preparing study sets to be loaned out. That is still under way, and we now have about one-third of the sets completed. We have them here for demonstration if you wish to see them. This will consist of 100 specimens that have been mounted and described fully. These are available for loan to any member of the Academy without charge, for use in study at home. There will be a small deposit, probably $15.00, required to cover possible breakages, but this will be returned upon return of the study set. The set costs us $2.25 a slide for the preparation of material. This is being given by the Academy in their donation last year of $500.00, and this year $500.00, and we trust that generosity will continue in the future. We expect that in about one year these sets will be available to any member of the Academy who wishes them for home study for a period of two or three months at a time.

One further plea: We need more material to complete certain specimens in the sets. This material can come only from the active members who contribute their material to the Museum. The man with a pathologic laboratory at his disposal will naturally not use the Museum, but the man who is not connected with a laboratory would best send his specimens to the Army Medical Museum. Put them in 1 per cent formalin, and ship them direct to the Army Medical Museum, Washington, D. C., and send with

the specimen as complete a case history or description as you can. The specimen will then be sectioned, studied, examined, and a report of the examination sent to you, all without charge. Furthermore, one or more sections of the specimen will also be sent to you, and if you desire to have photographs, they can be obtained at a minimum cost of something like twenty-five cents a piece. This is entirely without charge. It enriches the Museum, and gives you a permanent record of material which in the past has usually been consigned to the waste basket.

H. S. GRADLE.

REPORT OF THE AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY.

The American Committee on Optics and Visual Physiology has held two meetings in the past year. At the meeting of the American Medical Association in Minneapolis, the Chairman, Dr. Jackson, presented a report calling attention to the fact, that the bill in regard to "Optical Practitioners" presented to the British Parliament, had been rejected as inadvisable at this time.

He further called attention to the work of the Guild of Prescription Opticians of America. He urged ophthalmologists to cooperate with the Guild, which is composed of an ethical group of opticians, who are willing to serve the best interests of the public as we see them.

Our Committee at this meeting passed favorably on standards for admission of pupils to sight saving classes suggested by the National Society for the Prevention of Blindness. The standards are as follows:

RECOMMENDED IDEAL BASIS FOR ADMISSION OF PUPILS TO SIGHT SAVING CLASSES.

1. General statement. Children having a visual acuity of 20/70 or less in the better eye, after proper refraction. In addition, the following are recommended as potential candidates:

a. Children in elementary schools having four or more diopters of myopia;

b. Inactive, subsiding (or regressive) cases, such as interstitial or phlyctenular keratitis, optic neuritis, trachoma, etc., in which some irritation may be present, provided the approval of the attending physician is given.

2. All cases must be considered individually.

3. Any child who in the opinion of the ophthalmologist would benefit by assignment to a sight saving class, subject to suggestion for treatment and training by such ophthalmologist, and the acceptance of the educational authorities having charge of such classes.

4. It is assumed that all the children assigned to sight saving classes have average normal mentality.

WILLIAM H. WILDER, CHAIRMAN.

On motion of DR. CONRAD BERENS, seconded by DR. HORACE NEWHART, this report with its recommendations was adopted.

REPORT OF COMMITTEE ON LYE LEGISLATION.

Federal legislation to safeguard the distribution of packages of caustic poisons suitable for household use was passed on March 4, 1927, the Act known as Federal Caustic Poison Act.

The passage of the Federal Act does not do away with the need for State legislation. The Federal Act covers only interstate and foreign commerce and commerce within jurisdictions under the exclusive control of the Federal Government. Other commerce can be controlled only thru State legislation.

Legislation, in terms substantially identical with the terms of the Federal Caustic Poison Act, has been enacted in twelve States, and has received sensible construction in those States. The construction placed on the Federal Caustic Poison Act by the Department of Agriculture can hardly be so described; it is basically vague and broad, and it will apparently necessitate either court action to procure a reasonable construction of the law, or else amendment by Congress. It is likely that an appeal will be made to Congress.

The statute as passed by Congress covers only packages "suitable for household use." The Act authorizes the Secretary of Agriculture to make regulations to carry its provisions into effect. A regulation made by the Secretary defines the words "suitable for household use" to mean and imply "suitability for ready or convenient handling in places where people dwell." The question of the suitability of a package for household use is thus made to depend upon the ability of one or more members of the household conveniently to handle it. It does not depend on the form of the package (except as its form may prevent convenient handling) or on its contents. The definition disregards entirely the question whether the contents of the package are adapted to

any "household use" whatever, as that term is ordinarily understood.

An effort will be made to procure amendatory legislation from Congress during its next session that will correct the situation that has been created by the Department of Agriculture, and it will be necessary for those who desire to see an effective law remain on the statute books to be on their guard, lest the necessity for such amendments be seized on by interested parties as a vehicle for carrying thru amendments that may emasculate the Act.

The number of States named above as having adopted caustic poison legislation substantially identical with the Federal Act does not include States that have adopted such legislation in a more or less modified form. The total number of States now having legislation of this general type is nineteen, of which twelve have followed very closely the model State law.

Your Committee asks that the members in the different parts of the country be on their guard, and particularly do we ask the Publicity Committee, if they see anything of the kind in the press, to give us ample notice so that the various members of this Committee in the country at large may be on their guard to see that this Act is not repealed.

JOSEPH C. BECK, CHAIRMAN.

There being no objection, this report was received and filed.

REPORT OF THE COMMITTEE ON THE HARD OF HEARING.

Your Committee during the past year has been active chiefly in investigating and studying some of the underlying problems relating to the hard of hearing, in creating increased interest in these problems among the medical profession and educators, and in encouraging surveys to determine the incidence, degree and cause of hearing defects among children of school age.

Thru its individual members, who have contacts with the committee of other national groups of otologists, educators and social workers, it has kept in close touch with and has cooperated in the efforts of others likewise interested in the prevention of deafness and the conservation of hearing. There has recently developed a widespread interest and activity along these lines, as evidenced by a rapid increase in the number of cities which have officially adopted a system of periodic examination of the hearing acuity of school children, with the aid of the audiometer.

One of the members of your Committee has published the results of a survey of the schools for the deaf in the United States. Others have made numerous addresses before medical and educational groups, some of national scope, on the problem of the deafened. Your Society has been represented by several members in the important conferences on the Deaf and Hard of Hearing called by the Division of Anthropology and Psychology of the National Research Council. One of your Committee presented before the First International Otolaryngological Congress in Copenhagen, in August, 1928, the distinctly American application of the multiple telephone audiometer in testing large groups of school children.

Your Committee earnestly recommends:

1. That individual members of this Society whose practice includes otology actively interest themselves in behalf of the deafened and in all legitimate efforts in the prevention of deafness in their home communities, in order to counteract a growing feeling among an enlightened public, that the otologist is either not informed as to the large possibilities in deafness prevention and the improvement in the condition of the deafened, or is still complacently indifferent to the acute demands of a situation which has long existed, but which unfortunately the medical profession until recently has been unable to materially change. Unless we of the medical profession promptly rise to the occasion and exercise the leadership which is expected of us, there is grave danger that it will fall into less worthy hands.

2. Your Committee recommends that the Academy encourage research, particularly in the field of clinical otology, which with our new methods of approach promises to yield quick and far reaching results.

3. Your Committee feels that the time is near at hand for the passage in our several States of a uniform law, which shall effectively provide for the regular yearly testing of the acuity of hearing of all school children, by such standardized methods as shall be adequate to detect even slight degrees of hearing loss, as an initial step in the prevention of deafness and the conservation of hearing.

4. It is requested that this Committee be continued with the

power to augment its personnel by the election of other members subject to the approval of the President.

Respectfully submitted,

HOWARD V. DUTROW
HAROLD HAYS
CHARLES W. RICHARDSON
GEORGE L. TOBEY
CLAUDE T. UREN
HORACE NEWHART, CHAIRMAN.

There being no objection, this report was received and filed, the suggestions of the Committee to receive consideration.

REPORT OF YEAR BOOK COMMITTEE.

Your Committee on Year Book, in conjunction with that committee from the American Ophthalmological Society, and one from the Section on Ophthalmology of the American Medical Association, have held repeated meetings, and beg to report as follows:

It seems that the Year Book, in its past form should be entirely abandoned. The American Medical Association is taking over the Archives of Ophthalmology, which will be continued under the editorship of Dr. Knapp, and included in that will be a Year Book in modified form. Therefore, starting with the first of the year, the Year Book will again be available to you, in modified form, it is true, but we trust with the same excellence as that furnished by Dr. Jackson in the past.

Your Committee therefore feels that its function has been fulfilled and asks to be discharged.

H. S. GRADLE, CHAIRMAN.

REPORT OF COMMITTEE TO INVESTIGATE THE TREATMENT OF INCIPIENT CATARACT.

Your Committee wishes to report progress. We consider that the efforts ophthalmic surgeons are making to effect some influence on the progression of lenticular opacities are justified, especially those directed toward securing accurate refraction and the improvement of the patient's general health.

Two of your Committee are investigating lenticular opacities by direct photography. A well known firm of chemists has generously supplied a sufficient amount of expensive material to one of your Committee so that he can investigate the action of the substance on lenticular opacities, checked by direct photography and other scientific means.

Your Committee therefore wishes to be continued until all our available experiments can be tabulated and studied.

ALLEN GREENWOOD, CHAIRMAN.

There being no objection, the report was received and the Committee continued.

REPORT OF SECTION ON OPHTHALMOLOGY.

The Secretary for Ophthalmology wishes to express his appreciation of the kindly response to his invitation to open discussion on papers. Many of the papers on ophthalmology submitted on this program were prepared by special request, and in the aggregate, with the discussion, constitute an expression of current thought and practice among American ophthalmologists. The Academy is indebted to Dr. McDonald for the able discussion of an assigned subject prepared during the press of his other researches and undertaken upon the urgent request of his friends.

The Secretary would urge upon the membership, particularly the younger practitioners and newer members, the desirability of submitting papers on unusual cases and subjects of current interest. The program would be greatly enriched by a variety of contributions, discussed by men of wide experience.

W. L. BENEDICT, SECRETARY.

REPORT OF SECTION ON OTOLARYNGOLOGY.

The work of getting together the material for the program of the Section on Laryngology was commenced last Fall at the meeting in Detroit, by requesting members of the Academy to send in suggestions and subjects for papers which, in their opinion, would deepen the interest in our meetings. Request was made, also, that members send in the subjects on which they individually would like to present papers. The Secretary wrote personal letters to about forty of our members, asking them to send subjects for papers which they thought would be helpful to our members. We are grateful for the many replies received and for the helpful suggestions.

At the meeting of the Program Committee in St. Louis in December, 1927, all this accumulated material was presented, together with the names of members who had requested places on the program in 1928, with the subjects of the papers they wished to present. The Committee carefully considered the subject of each member who wished a place on the program, and would have

been pleased to gratify each man's request. But, as you know, since we have instituted the Postgraduate Course, which is proving to be so popular and profitable, we are having less than twelve prepared papers; therefore, some of our members had to be disappointed. We regret this very much and again earnestly appeal to the members to continue sending in requests and making suggestions.

Building a program, as all of you who have done that work know, is not an easy task. Much has to be taken into consideration. For example, has the subject been presented recently? Will it interest and help the greatest number of people? If it is case reports, have enough been studied to come to definite conclusions?

At the meeting in December, the program was outlined virtually as you have it, with only a few minor changes. The Secretary was instructed to secure four men to prepare five minute discussions of each paper, and we endeavored to have these discussions delivered personally by the writer. This plan has not worked out as desired, because some men have been kept away who had planned to come to the meeting when the program was in formation. It was also our plan not to have the same man on the prepared program more than once, thus assuring many studied opinions to be published in our Transactions, in order to make the Transactions better reference material. To the prepared discussions, of course, there will be added the extemporaneous discussions, all of which will make the volume more valuable.

One of the difficulties in our work was that of getting essayists to prepare their papers in time for the Presession Volume, published for the first time this year. Three papers, as you have noticed, we failed to have published in the Presession Volume. We regret this very much and hope that next year the essayists will so adjust themselves that all papers will be in the Presession Volume. We hope next year to have this volume issued in time for every member to read each paper, and thereby be better prepared to discuss the various papers in a satisfactory manner.

For next year's program we earnestly solicit (1) criticism of a constructive nature, (2) suggestions of subjects for papers that you think would be instructive, (3) requests for a place on the program.

We also ask that you ever bear in mind that the Program

Committee will do its utmost to prepare the best program possible from the material at hand.

JOHN MYERS, SECRETARY.

REPORT OF SECTION ON INSTRUCTION.

Your Committee presents its report Wednesday, Thursday and Friday mornings from nine to twelve-thirty. The work of the past year is shown those three mornings.

Your Committee wishes to carry out the Section on Instruction in accordance with the wishes of the majority of the Academy. We cannot get these wishes by telepathic communications, and we therefore ask for such criticism, constructive and destructive, as you may have to offer of our program.

We hope to continue next year, with one major change. In view of the rather important place that golf has assumed, and in view of the large number of people attending the ophthalmic and otolaryngologic examinations, Monday is fairly well taken up for those who are here. Many do not wish to arrive until Tuesday morning, consequently your Committee feels that registration for the course next year should not be held until Tuesday morning. We will then ask you to make your choice of courses, which will be numbered consecutively, to make your choice easier. We hope in this way to obviate the long wait which under our present system seems inevitable.

H. S. GRADLE, SECRETARY.

REPORT OF COMMITTEE ON NECROLOGY.

NELSON MILES BLACK, Necrologist: It is requested that those present rise while the names of members who have passed away during the past year are read:

Dr. Joseph Zepher Bergeron
Dr. Edward Leonard Bull
Dr. Walter Almond Hager
Dr. Edward Henry Higbee, Jr.
Dr. Frederick William Lamb
Dr. Henry Hager Martin
Dr. Frank Athon Morrison
Dr. Richard H. Satterlee
Dr. Greenfield Sluder
Dr. Herbert G. Tureman
Dr. Leon Edward White.

Joseph Zepher Bergeron

Dr. Bergeron was for a good many years associated in the practice of Dr. E. Fletcher Ingalls, doing nose and throat as well as chest work, and doing special work in climatologic therapy. Doctor Bergeron enjoyed a splendid high class practice in Chicago and was quite successful in results obtained for his patients, who always spoke very highly of him.

He taught at the Rush Medical College, was associated with the Dispensary there, and for one year was Associate Professor in Otolaryngology at Loyola University. In later years, he did surgery at the St. Joseph's Hospital. He enjoyed the friendship of his colleagues in Chicago.

As a contributor of medical literature he was not prolific, about the only contribution that he made to medicine, being the devising of an instrument to effect hemostasis of the tonsillar fossa.

Edward Henry Higbee

He was born in Whitehall, Illinois, on September 17, 1874. He was graduated from the Missouri Medical College in St. Louis, in 1897. Tho not an extensive writer, he was a very active member of the St. Louis Ophthalmic Society and a regular contributor to the program, with papers and discussions. Much industrial work was handled by him, and he was regarded as an authority on treatment of injuries to the eyes. He was a member of the American Academy of Ophthalmology and Oto-Laryngology and was on the staffs of many hospitals, including the Mullanphy, St. Louis City, Maternity and Isolation and City Sanitarium, St. John's and St. Anthony's. A man of wide acquaintance, a keen participant in sports, especially golf and hunting, a most agreeable companion in work or play; he is greatly missed in the places that know him no more. He died on September 20, 1927, at St. Luke's Hospital, St. Louis, of pneumonia.

Frederick William Lamb

Ophthalmologist, Rhinologist, Otolaryngologist, b. Maywood, Ill., Aug. 24, 1874, s. George Clark and Mary Louise (Heady).

Ed. Oberlin Coll., A.B. Beta Theta Pi, m. New York, N. Y. Jan. 29, 1913, Ninon Traver. Interne, Cincinnati General Hospital, 1899-1901, Clin. Prof. of Ophthalmology, Med. Dept. Univ. Cincinnati, Visiting Ophthalmologist, Cincinnati Gen. Hosp., Visiting Ophthalmologist, Christ Hosp., Visiting Ophthal., Chs. Hosp., Visiting Laryngol., Chs. Hosp., Cons. Ophthalmolog., Vet. Bur. Hosp. No. 69. Affr. in charge Phys. Exam. Unit, Air Serv.,

U. S. A., 1918. Mem. Med. Advisory Bd., Draft Service, U. S. A., 1918. Mem. Cincinnati Acad. Med., O. State Med. Assn., A.M.A., Am. Acad. Ophthal. and Oto-Laryngol., F.A.C.S.

Recreations: Golf, billiards. Clubs: Queen City, Cincinnati Country, Business Men's, Cincinnati Automobile. Rep. Prot.

Henry Hager Martin

He was born in Ashland, Kentucky, May 28th, 1866. He graduated from the Miami Medical College, Cincinnati, Ohio, 1891. He served interneship in the Cincinnati General Hospital, later going to Ashland, Ky., where he did general practice of medicine until 1898. That same year he went to New York City to enlist in the Spanish-American War, but was turned down on account of his physical disability. He then entered the Manhattan Ear, Eye, Nose and Throat Hospital in New York. He came to Savannah in 1900, with a patient suffering from carcinoma of the throat and was his private physician for six months.

He liked Savannah so well he opened an office and specialized in Diseases of the Ear, Eye, Nose and Throat, and continued to make Savannah his home until his death June 12th, 1928.

In 1906, he was elected President of the Georgia Medical Society. During the same year he helped to organize the Southern Medical Association and was its first President.

In 1915, he had the misfortune to be injured in an automobile accident which disabled him for two years, but during this time he was actively interested in the progress of the Southern Medical Association and its Journal.

In 1917, he offered his services to the United States Government during the World War, asking to be actively engaged in some capacity, later being appointed as Medical Advisor in the Selective Service System. For his services he was awarded a Service Medal.

During the last years of his practice he took a special interest in injecting the fifth nerve with alcohol, for tic douloureux.

October 19th, 1922, he was married to Sena Inez Bishop of Savannah, Georgia. There were no children.

He was a member of the A. M. A., the Triological Society and the American Academy of Oph. and O.-L., and was the author of many medical papers.

Richard H. Satterlee

He was for many years a well known ophthalmologist, and died at his family home on January 18th, 1928, after a brief illness from pneumonia.

Born in Rochester, N. Y., in 1861, he was the son of LeRoy Satterlee, president of the Collegiate Institute of New York. He attended the University of Buffalo and graduated from the Medical Department in 1888. After his graduation, he located in Easton, Pennsylvania, where he engaged in the practice of general medicine until 1894, when he took a special course in ophthalmology at the Manhattan Eye and Ear Hospital in New York, afterwards studying his specialty in London, Paris, and Vienna. He was three times married, his last wife being Miss Pearl Murphy of Shelby, New York.

He was a member of the Erie County and the New York State Medical Societies, of the American Medical Association, and of this Academy, also of the Buffalo Ophthalmological Club. He was not fond of surgery and was chiefly known as a skilled refractionist. He was of a retiring disposition and took part in medical discussions only when circumstances brought him out.

Herbert Gregory Tureman

Dr. Tureman was born in Callaway County, Missouri, "the Kingdom of Callaway," in 1875, and died at his home January 1, 1928, on the anniversary of his father's birth. His father was Col. Robert W. Tureman, a pioneer Missourian who came to Kansas City in 1885.

Dr. Tureman was a student at the Missouri Military Academy at Mexico, Mo., and graduated from the University Medical College, Kansas City, in 1897.

A few years was devoted to general practice in which his genial manner and self confidence impressed his patients, who were already numerous. Two years were then spent in hospitals in New York developing his specialty of laryngology and otology. This work was followed by postgraduate work in Berlin and Vienna for three years.

While a student abroad he met and was married to Miss Marie Powers, a student of piano under Godowsky and the late master, Scharwenka.

Dr. Tureman's professional affiliations were many, including membership in the Jackson County Medical Society, the Missouri State Medical Association, the American Medical Association, American Academy of Ophthalmology and Oto-Laryngology, the K. C. Eye, Ear, Nose and Throat Society, K. C. Academy of Medicine, and he was a Fellow of the American College of Surgeons. He was a member of the Staffs of St. Luke's Hos-

pital, Trinity Lutheran Hospital, Research Hospital and St. Joseph Hospital.

Dr. Tureman enjoyed many activities outside his professional work, being a director of the Missouri Savings and Trust Company, member of the Kansas City Country Club, the Mission Hills Country Club, the University Club, the Kansas City Club, two gun and hunting clubs, and several Masonic bodies, and was a communicant of the Westminster Congregational Church.

Leon E. White

A specialist in diseases of the ear, nose and throat, in Boston, died of coronary sclerosis, a form of heart disease, at his home at 112 Wolcott Road, Friday, May 18.

He was born in Barton, July 30, 1867, the son of Orra A. and Emily B. (King) White. He graduated from Barton Academy in the first class ever graduated. He graduated from St. Johnsbury Academy in 1886, and from Dartmouth College in 1890, and followed that with a medical course in the same college. He took many prizes and honors thruout his educational course, winning the highest standing any man had taken in Dartmouth Medical School in ten years. After a few years as a general practitioner, he became house surgeon at the Massachusetts Charitable Eye and Ear Infirmary, and continued as assistant surgeon for a number of years following. For several years he was surgeon in Otology at St. Elizabeth's Hospital. He was a member of the American Medical Association, American Board of Otolaryngology, American Otological Society, American Laryngological Society, American Academy of Ophthalmology and Oto-Laryngology, American Rhinological and Otological Society and Fellow in Otology, Harvard Postgraduate School. He became nationally known in his profession, and was frequently called to the large cities of this country and Canada to read papers or deliver lectures before medical societies. About two weeks before he died, he went to Washington, D. C., to present a paper to a gathering of specialists.

He was a man of magnetic personality and of sterling worth, drawing around him a large circle of friends who appreciated his noble and manly qualities. He was a tower of strength to everyone who came into his benevolent presence, and a more beloved man by patients and physicians can never be found.

While a hard worker and entirely devoted to his profession, he found time for the enjoyment of all that was beautiful in nature. Born among the Vermont hills, he always had a very

keen love for birds, flowers, the great mountains and the open spaces. He made four trips across the country to visit the most celebrated features of American scenery. He loved the simple life and was devoted to his home.

In the death of Dr. Leon E. White, there has been removed from the profession, one of its most able members.

When one looks over the long list of papers which he has written on various subjects, beginning with the year 1899, he is impressed with the versatility of his interests. He was one of the very first to suggest and to improve the operation for the resection of the nasal septum; after that he began his work on retrobulbar neuritis, and there is a list of twenty-three articles written by him on that most important subject. Can one render any greater service to humanity, than the cure of blindness and the preservation of sight?

Election of Members.

The Secretary read the following list of candidates whose names had been passed upon favorably by the Council:

Adams, Charles Joseph, 618 A. and L. Bldg., Kokomo, Indiana, OALR.

Allen, Orris T., 422 Rose Dispensary Bldg., Terre Haute, Indiana, OALR.

Allen, Thomas Dyer, 122 S. Michigan Ave., Chicago, Op.

Armstrong, R. M., 1010 Fayette Bank Bldg., Lexington Ky., OALR.

Begle, Howell Llewellyn, 1513 Longfellow Ave., Detroit, Mich., Op.

Bell, Will Otto, 1431 Medical-Dental Bldg., Seattle, Wash., Op.

Blair, Mortimer Warren, N. E. cor. Chestnut and 20th, Phila., Pa., Op.

Breitenbach, Oscar C., 614 Waukegan Nat. Bank Bldg., Waukegan, Ill., OALR.

Brownell, Morton Emmons, 1019 First Nat. Bk. Bldg., Wichita, Kan., Op.

Bryan, James Leslie, 919 Bennie-Dillon Bldg., Nashville, Tenn., OALR.

Bulson, Eugene Loring, 406 W. Berry St., Fort Wayne, Indiana, OALR.

Carruth, Howard Ernest, 716 Medical Arts Bldg., Portland, Oregon, OALR.

Cassady, J. Vernal, 654 Associates Bldg., South Bend, Indiana, OALR.
Christoph, Carl H., 30 N. Michigan Ave., Chicago, OALR.
Cody, Claude Carr, 1306 Walker Ave., Houston, Texas, ALR.
Cowan, Alfred, 2007 Pine St., Philadelphia, Pa., Op.
Crumrine, Norman Ross, Colonial Apts., Beaver, Pa., OALR.
Cunning, Daniel S., 114 E. 54th St., New York City, OALR.
Darrow, C. H., 110 Metropolitan Bldg., Denver, Colorado, ALR.
Davis, Charles L., 24 S. Gore Ave., Webster Groves, Mo., ALR.
Davis, John Calvin, Jr., 425 Aquila Court, Omaha, Nebraska, OALR.
Deal, John Francis, First Natl. Bank Bldg., Springfield, Ill., OALR.
Delph, John Francis, 104 S. Michigan Blvd., Chicago, ALR.
DeWitt, Edward Nicholas, 836 Myrtle Ave., Bridgeport, Conn., Op.A.
Dickson, John Forest, 1104 Selling Bldg., Portland, Oregon, OALR.
Dixon, Otto Jason, 902 Medical Arts Bldg., Kansas City, Mo., ALR.
Earley, James Hugh, 1613 Connecticut Ave., Washington, D. C., ALR.
Eber, Carl Theodore, 1000 Carleton Bldg., St. Louis, Mo., Op.
Egan, Burton Wright, 408 North St., Logansport, Indiana, OALR.
Eimer, Charles Eugene, 316 Lister Bldg., St. Louis, Mo., ALR.
Ellis, Charles Ward, Lansing, Michigan, ALR.
Eubank, Ambrose E., 636 Argyle Bldg., Kansas City, Mo., OALR.
Farrior, J. Brown, 605 Citizens Bank Bldg., Tampa, Fla., OALR.
Fernlund, George Byron, 33 Montrose Court, Johnson City, Tenn., OALR.
Ferris, David P., 1000 Carleton Bldg., St. Louis, Mo., ALR.
Folk, Max Lyon, 55 E. Washington St., Chicago, OALR.
Folken, Frank Garrett, 306 Albert Lea State Bank Bldg., Albert Lea, Minn., OALR.
Ford, Hanby Lewis, 401 Robeson Bldg., Champaign, Ill., OALR.
Frankel, Edward, Jr., 152 W. 58th St., New York City, ALR.
Frost, Albert Dalbey, 681 E. Broad St., Columbus, Ohio, Op.
Frothingham, Edward, 8644 94th St., Woodhaven, N. Y., OALR.
Fuchs, Valentine Henry, 220 Chaille Bldg., New Orleans, La., ALR.

Fuller, John Andre, Reno, Nevada, OALR.
Gaudet, Lucien Sydney, 405 Franklin St., Natchez, Miss., OALR.
Gill, Arthur Ernest, U. S. Veterans' Hosp. No. 88, Memphis, Tenn., OALR.
Glick, Harry Naylor, 3102 S. Grand Ave., St. Louis, Mo., ALR.
Glowacki, Ben Francis, 770 Maccabee Bldg., Detroit, Mich., OALR.
Goldman, A. Milton, 121 E. 60th St., New York City, Op.
Greene, Laurence Whitridge, 806 Metropolitan Bldg., Denver, Col., ALR.
Greenfield, William John, The Polyclinic, 20 S. Dunlap St., Memphis, Tenn., ALR.
Hale, Arthur Stanley, 3790 Woodward Ave., Detroit, Mich., OALR.
Hall, Edwin Perry, 936 University Bldg., Syracuse, N. Y., ALR.
Hardy, LeGrand Haven, 30 E. 40th St., New York City, Op.
Harrison, Fred Hazard, 410 Medical Arts Bldg., Portland, Oregon, OALR.
Hill, Frederick Thayer, Professional Bldg., Waterville, Maine, ALR.
Hill, Howard Foster, Professional Bldg., Waterville, Maine, Op.
Hughes, Ray Whitbeck, 1501 David Whitney Bldg., Detroit, Mich., OALR.
Hutchinson, Abbott Trask, 30 W. 59th St., New York City, ALR.
Jackson, Atras Earl, Medical Arts Bldg., Fort Worth, Texas, Op.
Jaquish, Charles Joseph, 904 Westinghouse Bldg., Pittsburgh, Pa., ALR.
Jones, Cecil Cain, 1120 Equitable Bldg., Des Moines, Iowa, OALR.
Jones, Edley Hicks, Vicksburg, Miss., OALR.
Jones, Vincent L., 3511 Washington St., St. Louis, Mo., Op.
Karkett, Robert Budd, 1203 Selling Bldg., Portland, Oregon, OALR.
Kelley, Isaac D., Jr., 801 Beaumont Medical Bldg., St. Louis, Mo., ALR.
Kelly, Joseph Dominic, 114 E. 54th St., New York City, OALR.
Kennedy, Eldred Weston, 282 Alexander St., Rochester, N. Y., Op.
Killeen, John Joseph, 104 S. Michigan Ave., Chicago, ALR.

Kincannon, Leroy Taylor, The Woodward, Birmingham, Alabama, OALR.
Kinne, Sandford Halsey, 73 Main St., Binghamton, N. Y., ALR.
Koenig, Ivan J., 40 North St., Buffalo, N. Y., Op.
Krimsky, Joseph, 263 Eastern Parkway, Brooklyn, N. Y., OALR.
Kuhn, Hugh A., 686 Hohman Bldg., Hammond, Indiana, ALR.
Lawwill, Stewart, 801 Chattanooga Bank Bldg., Chattanooga, Tenn., OALR.
Lewis, Philip Meriwether, Exchange Bldg., Memphis, Tenn., OALR.
Lierle, Dean McAllister, Burkley Place, Iowa City, Iowa, OALR.
Lucas, Harold Roy, 411 Selling Bldg., Portland, Oregon, OALR.
Lupton, Irving Martin, Medical Arts Bldg., Portland, Oregon, OALR.
Mahoney, Paul Leo, Donaghey Bldg., Little Rock, Arkansas, ALR.
Marks, Samuel Blackburn, Sec. Trust Bldg., Lexington, Ky., ALR.
Mason, Roy Earl, 422 Frisco Bldg., St. Louis, Mo., Op.
Masters, Robert John, 601 Hume-Mansure Bldg., Indianapolis, Indiana, Op.
McKee, Joseph Wallace, 1222 Rialto Bldg., Kansas City, Mo., Op.
McKee, Otto S., 131 Linwood Ave., Buffalo, N. Y., ALR.
McNally, William James, 746 Sherbrooke St., West, Montreal, Canada, ALR.
Merkel, Charles Carson, 1409 David Whitney Bldg., Detroit, Mich., ALR.
Mertins, Paul Stearns, 1115 Bell Bldg., Montgomery, Alabama, OALR.
Meyer, Samuel James, 58 E. Washington St., Chicago, Op.
Montgomery, Daniel Cameron, Greenville, Miss., ALR.
Moore, Roy Seeley, 405 E. Fayette St., Syracuse, N. Y., ALR.
Moriarty, Richard W., 38 S. Broadway, White Plains, N. Y., OALR.
Neumann, William Henry, 1005 N. 8th St., Sheboygan, Wis., OALR.
Palmer, Bascom Headen, 502 Huntington Bldg., Miami, Fla., OALR.
Palmer, Frank Ernest, Sterling, Colorado, OALR.
Piper, Arthur Sylvester, Enid Clinic, Enid, Oklahoma, OALR.
Plumer, John Shaffer, 121 University Place, Pittsburgh, Pa., Op.

Poos, Grover Hewitt, 301 University Club Bldg., St. Louis, Mo., OALR.

Powell, Lyle Stephenson, 813 Mass. St., Lawrence, Kansas, OALR.

Prince, Alexander L., 36 Pearl St., Hartford, Conn., Op.

Qualls, Harley Winfield, 1501 Exchange Bldg., Memphis, Tenn., OALR.

Randall, Hiram, 81 Main St., Binghamton, N. Y., ALR.

Reaves, Robert Grisham, Knoxville, Tenn., OALR.

Reese, Algernon B., 50 W. 52nd St., New York City, Op.

Rhode, Solon Lafayette, 236 N. 6th St., Reading, Pa., OALR.

Risser, Edward D., 741 Winona St., Winona, Minn., OALR.

Rogers, Robert Merriam, 680 St. Johns Place, Brooklyn, N. Y., Op.

Rychener, Ralph Orlando, 1720 Exchange Bldg., Memphis, Tenn., Op.

Sanor, Daniel Galbreath, 206 E. State St., Columbus, Ohio, ALR.

Schneider, Anton S., 56 Court St., Plattsburg, N. Y., Op. A.

Schwartz, William A., Phoenix, Arizona, OALR.

Senseney, Eugene Towner, 316 Lister Bldg., St. Louis, Mo., ALR.

Sinamark, Andrew, 205 Power Bldg., Hibbing, Minn., OALR.

Sisson, Raymond J., 753 David Whitney Bldg., Detroit, Mich., Op.

Smit, William Maurice, 719 Missouri Theatre Bldg., St. Louis, Mo., ALR.

Snapp, Carl Foster, 604 Medical Arts Bldg., Grand Rapids, Mich., ALR.

Spaeth, Edmund B., N. E. cor. Chestnut and 20th, Philadelphia, Pa., Op.

Spencer, George Albert, 1005 Seventh St., Sacramento, Calif., OALR.

Sproule, Ralph P., 141 E. Wisconsin St., Milwaukee, Wis., OALR.

Stanton, Harmon Lamontte, Cleveland Life Bldg., Evansville, Indiana, OALR.

Steele, Willard, Provident Bldg., Chattanooga, Tenn., OALR.

Stinson, William David, 805 Medical Arts Bldg., Memphis, Tenn., ALR.

Swab, Charles Marion, Medical Arts Bldg., Omaha, Nebraska, Op.

Teall, Raymond Edwin, 1100 Rialto Bldg., Kansas City, Mo., ALR.

Thomas, James W., 726 Security Mutual Bldg., Lincoln, Nebraska, OALR.

Thompson, Howard E., 1100 Main St., Dubuque, Iowa, OALR.

Walker, Waldo Webster, 270 Commonwealth Ave., Boston, Mass., ALR.

Warren, William C., Jr., 618 Adams Nat. Bank Bldg., Atlanta, Ga., ALR.

Warwick, Harold Lynwood, 913 Medical Arts Bldg., Fort Worth, Texas, OALR.

Weber, Henry Charles, U. S. Naval Hospital, Great Lakes, Ill., OALR.

Weinberger, Nelson S., Robert Packer Hospital, Sayre, Pa., OALR.

Whalen, Edward Joseph, 750 Main St., Hartford, Conn., ALR.

Willson, Wesley W., 563 David Whitney Bldg., Detroit, Mich., OALR.

Windham, Robert Edward, Longview, Wash., OALR.

White, William Beverly, 813 Gurley Bldg., Stamford, Conn., OALR.

Wood, Earl LeRoy, 33 Lincoln Park, Newark, N. J., ALR.

Wood, V. Visscher, 201 Humboldt Bldg., St. Louis, Mo., ALR.

Woodson, Burbank Palmer, Temple, Texas, OALR.

Yost, Walter Milton, Peoples Bank Bldg., Rochester, Pa., Op.

Zubak, Matthew Francis, 75 12th St., Wheeling, W. Va., OALR.

THE SECRETARY: During the last several months, in preparation for this very splendid meeting of the Academy, the members in St. Louis had the very laudable ambition to present to the Academy a class of applicants to be voted in at this meeting. Due to an oversight in the Secretary's office, the St. Louis membership was not advised of the thirty-day limit, and they, not thinking of it, did not send their list of new members in to the Secretary's office in time for the thirty-day publication. And, altho it is violating a part of our Constitution, the Council has seen fit to unanimously elect part of the St. Louis class who have been certificated at this time. This will need the unanimous vote of those present, and I may say that the St. Louis members have themselves given their consent.

DR. GEORGE M. COATES: I move that this action of the Council be ratified by the members of the Academy. Motion seconded and unanimously carried.

THE SECRETARY: I will read this list of seven names, which makes a total of 146 up for election:

Alvis, Bennett Y., Carleon Bldg.....St. Louis
Brown, Clyde O., 3904 Laclede Ave.....St. Louis
Calhoun, James G., Metropolitan Bldg.....St. Louis
Howard, Francis C., University Club Bldg.....St. Louis
Howard, Harvey J. .....St. Louis
Mayer, Leo L., 308 N. Sixth St.....St. Louis
Miller, Vernon B.....St. Louis

DR. ARTHUR J. BEDELL: I move that the men whose names have just been read by the Secretary be elected to membership. Motion seconded and carried.

THE PRESIDENT: We now come to the Election of Officers. The Secretary will read the list.

THE SECRETARY: The following officers have been proposed for election:

President	Harris P. Mosher	Boston
President-elect	William H. Wilder	Chicago
First Vice-president	Frank E. Burch	St. Paul
Second Vice-president	Harry W. Lyman	St. Louis
Third Vice-president	John H. Dunnington	New York
Treasurer	Secord H. Large	Cleveland
Editor of Transactions	Clarence Loeb	Hubbard Woods, Illinois
Executive Secretary	William P. Wherry	Omaha
Secretary for Ophthalmology	William L. Benedict	Rochester, Minn.
Secretary of Otolaryngology	John L. Myers	Kansas City, Mo.
Secretary for Instruction	Harry S. Gradle	Chicago
Member of Council	John J. Shea	Memphis, Tenn.
Member of Ophthalmological Board	John M. Wheeler	New York

Moved by DR. A. J. BEDELL that the Secretary cast the ballot of the Academy for these men as officers for the ensuing year. Motion seconded and carried, and the officers declared elected.

THE PRESIDENT: The last item of business is the selection of a place of meeting for 1929.

DR. HORACE NEWHART: I move that the Academy hold its next meeting in Atlantic City. Motion seconded and carried.

THE PRESIDENT: The Chair will appoint Dr. Greenwood and Dr. Large to escort the President-elect to the rostrum.

DR. WILLIAM H. WILDER: I am so unaccustomed to situations like this that I cannot find words to express my feelings. The surprise is indeed great, and I wish merely to assure you of my deep appreciation of the confidence and the honor you have bestowed upon me, and to say that I will do the best I can to uphold the fine traditions of this Society and of the men who have worked so ably for it in the past. I trust I may have the cooperation that my predecessors have had to help me carry on the work. The splendid spirit that is manifested in this Society, I think, will be ample inspiration to help me along with my task.

No further business appearing, the Business Meeting adjourned.

October 18, 1928.

The Thursday afternoon session was called to order at two o'clock, the First Vice-president, Dr. Frank L. Dennis, presiding.

THE CHAIRMAN: I would like to say a word about the Presession Volume. As Dr. Mosher emphasized at the President's Luncheon, the men will not get the best results from this volume until they learn to abstract their papers. As you know, the idea of the Presession Volume is to save time for what is regarded as the most valuable part of the meeting, namely, the discussion. When each man, in the first place, sees that his paper gets into the Presession Volume, and in the next place reads a short abstract bringing out the salient points, then there will be a few minutes for pictures that the essayists may wish to present. In that way, we will save time that may be devoted to discussion. This is simply a suggestion, that we may make better use of this method.

October 19, 1928.

The Friday afternoon session was called to order at two o'clock, Dr. Luther C. Peter, presiding.

The CHAIRMAN first introduced DR. F. E. HARRINGTON, Commissioner of Health and Director of Child Hygiene, Minneapolis, Minnesota, who showed lantern slides demonstrating the taking of vision of groups of school children.

DR. WALTER E. HOFFMAN, Seattle, extended an invitation to the members of the Academy to attend the meeting of the Pacific Coast Otolaryngological Society in Salt Lake City next spring—

either immediately preceding or following the meeting of the American Medical Association in Portland.

THE CHAIRMAN: The Chair is asked to announce that Professor Marx, who has a great deal of work on hand in connection with the International Congress of Ophthalmology which will meet next summer, finds it impossible to communicate with each one in regard to hotel accommodations, etc., and has delegated a good deal of this work to Thomas Cook and Sons, and the Academy has authorized Thomas Cook and Sons to be the official travel agency for this Society. Anyone who wishes to make hotel reservations can make them thru Cook and Sons.

THE PRESIDENT: This brings to a close the scientific part of the program. Before I complete the last official act of my office, I want to express to the membership and the officers, and to all who contributed to the making of this meeting a great success, my heartiest appreciation.

I have enjoyed this year very much. The last week has been rather strenuous, but it is worth it all, and I only hope that the incoming President will get as much pleasure out of presiding over this body as I have.

Dr. Mosher, President of the Academy, it is a great pleasure to me to hand over the gavel of authority, with my heartiest congratulations and best wishes for a successful year.

DR. HARRIS P. MOSHER, President American Academy of Ophthalmology and Oto-Laryngology: Mr. President, and Members of the Academy: In receiving this emblem of temporary authority, I appreciate very keenly the responsibility that goes with it. As one of the executives of this Academy for the next year, it will be our high ambition to repeat, if possible, the great success of the present meeting, which I now declare closed.

The American Academy of Ophthalmology and Oto-Laryngology adjourned *sine die*.

ALPHABETIC LIST

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Graves, Basil.....35 Weymouth St., W., London, Eng.
Gray, Albert A.....Glasgow, Scotland
Johnson, Lindsay,
Britannia Bldg., West St., Durban, Natal, Africa
Kirkpatrick, Lt.-Col. Henry,
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Lawford, J. B., 27 Weymouth St., Portland Pl., W., London, Eng.
Lister, Sir William T., 24 Devonshire Place, W. 1, London, Eng.
Nagar, Prof. F.....Freiestrasse 20, Zurich, Switzerland
Van der Hoeve, Prof. J...Rijnsburgerweg, 6 A, Leiden, Holland

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Standish, Myles,
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Stewart, T. M.....901 Union Trust Bldg., Cincinnati, O., OALR
Wood, Casey A.....7 W. Madison St., Chicago, Ill., OALR
Woods, Hiram.....842 Park Ave., Baltimore, Md., Op.A.

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* Attended 1928 meeting. Numbers in parentheses indicate year in which the Fellow became a member of the Academy. Follows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary, Dr. W. P. Wherry, Medical Arts Bldg., Omaha, Neb.

Fellows

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B

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BARKAN, ADOLPH . . Union Trust Bldg., San Francisco, Cal., OALR
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BEACH, B. S. (1927) . . . . . 76 W. 86th St., New York, N. Y., Op.
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BERNSTEIN, E. J. (1900) . . . . . 10 Peterboro, Detroit, Mich., OALR
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*BINKLEY, ROY S. (1915) . . . . 530 Fidelity Bldg., Dayton, O., OALR
*BIRSNER, LOUIS J. (1926)
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*BLAAUW, E. E. . . . . 190 Ashland Ave., Buffalo, N. Y., Op.
*BLACK, MELVILLE (1904)
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*BLACK, N. M. . . . 120 E. Wisconsin Ave., Milwaukee, Wis., Op.
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BLUM, HENRY,
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BOBB, E. V. (1918)
101 First Nat'l Bk. Bldg., Mitchell, S. D., OALR
BOECKMANN, EGIL. . . . . Lowry Bldg., St. Paul, Minn., OALR
BOEHRINGER, W. H.
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BOERNER, MORRIS H. (1923)
Scarbrough Bldg., Austin, Tex., OALR
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BONE, HERMAN D. . . . . 19 Pleasant St., Gardner, Mass., OALR
BONNER, WM. F. (1926)
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BRAY, E. R. (1912) . . . . . 342 Lowry Bldg., St. Paul, Minn., OALR
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*BRICKLEY, DANIEL W. (1918)
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*BRIGGS, H. H. . . . . 101 Haywood St., Asheville, N. C., OALR
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*BROWN, CLYDE O. (1928)
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BRUMM, SETH A. (1922)
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BRUNER, ABRAM BRENEMAN (1922)
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BUTLER, ROBERT H. (1920) . . . . . Bellefontaine, O., OALR

BUTT, MIRIAM M....2045 Chestnut St., Philadelphia, Pa., Op.
BUVINGER, CHARLES W.(1921)
50 Washington St., East Orange, N. J., OALR
BYRNES, H. F.....67 Chestnut St., Springfield, Mass., OALR

C

CAHILL, J. W.....390 Main St., Worcester, Mass., OALR
CALDWELL, ROBERT
831 New Donaghey Bldg., Little Rock, Ark., OALR
CALHOUN, F. PHINIZY(1911)
478 Peach Tree St., Atlanta, Ga., Op.
*CALHOUN, JAS. G.....Metropolitan Bldg., St. Louis, Mo., Op.
*CALLFAS, W. F.....1620 Med. Arts Bldg., Omaha, Neb., ALR
CAMERON, WM. G...Puget Sd. Bk. Bldg., Tacoma, Wash., OALR
*CAMP, W. E.(1920)
716 Donaldson Bldg., Minneapolis, Minn., OALR
CAMPBELL, C. H.
Columbus State Bank Bldg., Columbus, Neb., OALR
CAMPBELL, D. A.....David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, DON M.(1908)
David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, M. D.(1927)
David Whitney Bldg., Detroit, Mich., OALR
*CAMPBELL, ROBERT A.,
520 LaSalle Bldg., Minneapolis, Minn., OALR
CAMPBELL, W. K.....96 Third Ave., Long Branch, N. J., OALR
CAPRON, F. P.....118 Angell St., Providence, R. I., OALR
CARLETON, RALPH(1914) 10 Temple St., Springfield, Mass., Op.
*CARMACK, J. W.(1919)
505 Hume-Mansur Bldg., Indianapolis, Ind., ALR
*CARMODY, T. E.(1904)..Metropolitan Bldg., Denver, Colo., ALR
CARMODY, THOMAS J.(1926)
408 The Temple, Danville, Ill., OALR
CARNCROSS, HELEN(1920)
58 E. Washington St., Chicago, Ill., Op.
CARPENTER, E. R.....610 Marvin Bldg., Dallas, Tex., OALR
CARPENTER, E. W.(1920)...Prof. Bldg., Greenville, S. C., OALR
CARR, GEO. W.(1909) 54 S. Franklin St., Wilkes-Barre, Pa., OALR
CARROLL, GEO.G.....614 W. Main St., Rochester, N. Y., OALR
CARRUTH, HOWARD ERNEST(1928)
716 Med. Arts Bldg., Portland, Ore., OALR
*CARSON, W. E.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
CARTER, W. W.(1896) 2 E. 54th St., New York City, N. Y., ALR
CARVILLE, L. MAUD(1914)
82 Commonwealth Ave., Boston, Mass., Op.
*CARY, E. H.....Medical Arts Bldg., Dallas, Tex., OALR
CARY, G. C.....Grand Junction, Colo., OALR
*CASSADY, J. VERNAL(1928)
654 Associate Bldg., South Bend, Ind., OALR

CAVANAUGH, J. A. . . 1712-30 N. Mich. Blvd., Chicago, Ill., ALR
*CAYCE, E. B. (1912) . . . . Med. Arts Bldg., Nashville, Tenn., OALR
*CHADWICK, IRA B. (1920)
4 Terminal Bldg., Coffeyville, Kan., Op.
CHAPMAN, SOLOMON J. (1926)
402 Burns Bldg., Colorado Springs, Colo., ALR
CHAPMAN, V. A. . . . . 445 Milwaukee St., Milwaukee, Wis., OALR
CHAPPELL, R. S. (1925)
305 Traction Terminal Bldg., Indianapolis, Ind., ALR
*CHARLES, JOS. W. . . . 3720 Washington Ave., St. Louis, Mo., Op.
*CHARLTON, C. C. . 124 S. Illinois Ave., Atlantic City, N. J., ALR
*CHASE, E. FRANK (1918) 1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, S. B. (1925) . . . . . Carver Bldg., Ft. Dodge., Ia., OALR
CHASE, W. D. (1924) . . . . 230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. FRANKLIN . . . . . 671 Broad St., Newark, N. J., Op.
CHENERY, W. E. . . . 377 Commonwealth Ave., Boston, Mass., ALR
CHENEY, JAMES W. . . . 702 Schweiter Bldg., Wichita, Kan., OALR
*CHILDS, H. A. . . . . Eagle Blk., Creston, Ia., OALR
CHIPMAN, L. D. V. (1925)
42 Coburg St., St. John, N. B., Canada, OALR
CHRISTOPH, CARL H. (1928)
30 N. Michigan Ave., Chicago, Ill., OALR
*CLARK, H. S. (1916) . . . LaSalle Bldg., Minneapolis, Minn., OALR
*CLARK, I. G. . . . . 188 E. State St., Columbus, O., OALR
*CLARK, J. SHELDON (1914)
509 State Bk. Bldg., Freeport, Ill., OALR
CLARK, L. H. (1915) . . 337 Monroe Ave., Rochester, N. Y., OALR
*CLAY, G. E. (1917) . . . . . Med. Arts Bldg., Atlanta, Ga., Op.
CLEFF, OSCAR (1926) . . . 55 E. Washington St., Chicago, Ill., OALR
CLEMENT, C. C. . . . . 30 N. Michigan Blvd., Chicago, Ill., OALR
CLERF, LOUIS HENRY (1923)
128 S. 10th St., Philadelphia, Pa., ALR
*CLEVENGER, W. F. (1905)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
CLIFFORD, T. P. (1927)
1320 David Whitney Bldg., Detroit, Mich., OALR
CLOTHIER, JOSEPH . . . . . Pocatello, Ida., OALR
*COATES, GEO. M. (1922) . . . 1721 Pine St., Philadelphia, Pa., ALR
COBB, EDWIN (1912) . . Masonic Temple, Marshalltown, Ia., OALR
*CODY, CLAUDE CARR (1928)
1306 Walker Ave., Houston, Tex., ALR
COFFELT, THEODORE A.
508 Woodruff Bldg., Springfield, Mo., OALR
COGAN, J. E. . . . . 707 Rose Bldg., Cleveland, O., OALR
COHEN, HERMAN B. (1927)
Spruce & 13th St., Philadelphia, Pa., ALR
COHEN, MARTIN, 1 West 85th St., New York City, N. Y., Op.
COHEN, SAMUEL (1920) . . . . 2009 Pine St., Philadelphia Pa., ALR
COHN, FELIX . . . . . 47 W. 87th St., New York City, N. Y., ALR
COHN, SELDON . . . . . Fulton, Ky., OALR
COLLINS, E. W. . . . . 508 Majestic Bldg., Denver, Colo., ALR

COLLINS, J. D.(1914)..150 Main St., Northampton, Mass., ALR
COLVER, B. N.(1913)....Sanitarium 1, Battle Creek, Mich., ALR
COMEE, W. C.(1922)...Cady-Land Bldg.,Green Bay, Wis., OALR
CONLON, F. A.....301 Essex St., Lawrence, Mass., OALR
*CONNOR, CHAS. E.(1920)...Hamm Bldg., St. Paul, Minn., ALR
*CONNOR, RAY(1905).....1410 Stroh Bldg., Detroit, Mich., OALR
COOK, W. ALBERT(1924)...506 Palace Bldg., Tulsa, Okla., OALR
*COOPER, C. E.....652 Metropolitan Bldg., Denver, Colo., ALR
COOVER, D. H.(1918)
820 Metropolitan Bldg., Denver, Colo., Op.
*COPPS, LYMAN ALDEN(1923)
Marshfield Clinic, Marshfield, Wis., OALR
CORBETT, J. J.(1925).....520 Beacon St., Boston, Mass., OALR
*CORSER, J. B.....345 Wyoming Ave., Scranton, Pa., Op.
*COSTEN, J. B.(1925) 3720 Washington Ave., St. Louis, Mo., ALR
COTT, C. C.....483 Delaware Ave., Buffalo, N. Y., ALR
COTT, D. N.(1924)
141 St. Marks Pl., St. George, Staten Isl., New York, ALR
COULTER, CHAS. F.
404 1st Natl. Bk. Bldg., Great Falls, Mont., OALR
*COWAN, ALFRED(1928)...2007 Pine St., Philadelphia, Pa., Op.
*COWLEY, ROBERT HENRY(1926).....Berea, Ky., OALR
*COWPER, H. W.....543 Franklin St., Buffalo, N. Y., Op.
CRAFT, KENNETH L.(1920)
1003 Hume-Mansur Bldg., Indianapolis, Ind., ALR
CRANE, J. WELLINGTON(1921)
128 Third Ave., Trenton, N. J., OALR
CREIGHTON, WM. J.(1910)
258 S. 18th St., Philadelphia, Pa., Op.
*CRISP, WM. H.(1913)..Metropolitan Bldg., Denver, Colo., Op.
*CROCKETT, R. L...Madison Co. Trust Bldg., Oneida, N. Y., Op.
CROMAN, JOS. M., JR.(1924)
Washington Apts., Mount Clemens, Mich., OALR
CROSKEY, J. W.(1905)..Med. Arts Bldg., Philadelphia, Pa., Op.
CROSS, A. E.(1914).....390 Main St., Worcester, Mass., Op.
CROSS, G. H.(1919).....525 Welsh St., Chester, Pa., Op.
CROSSLEY, E. R.....30 N. Michigan Blvd., Chicago, Ill., OALR
*CRUMRINE, NORMAN ROSS(1928)
595 Third St., Beaver, Pa., OALR
CULBERTSON, L. R.(1898) Masonic Temple, Zanesville, O., OALR
CULLOM, M. M.(1910)
1119 Bennie-Dillon Bldg., Nashville, Tenn., OALR
CUNNING, DANIEL S.(1928)
114 E. 54th St., New York City, N. Y., OALR
CUNNINGHAM, ALLAN R.(1924)
260 Barrington St., Halifax, N. S., OALR
*CURDY, R. J.....1134 Rialto Bldg., Kansas City, Mo., Op.
CURRAN, E. J.(1919)
1805 Federal Reserve Bk. Bldg., Kansas City, Mo., Op.
CURRY, GLENDON E.(1912)
Westinghouse Bldg., Pittsburgh, Pa., Op.

CURTIN, EUGENE A.(1923)
703 Connell Bldg., Scranton, Pa., OALR
CURTIS, W. L.....1503 So. 22nd St., Lincoln, Neb., OALR
*CUSHMAN, BEULAH(1924)
25 E. Washington St., Chicago, Ill., Op.
CUTLER, FRANKLIN E..1026 Schofield Bldg., Cleveland, O., ALR

D

DABNEY, S. G.....Starks Bldg., Louisville, Ky., OALR
DABNEY, VIRGINIUS(1913)
1633 Connecticut Ave., Washington, D. C., ALR
*DAILY, LOUIS(1926) 1117 Med. Arts Bldg., Houston, Tex., ALR
*DAILY, RAY K.(1926)
1117 Med. Arts Bldg., Houston, Tex., OALR
*DANCY, ALEXANDER B.
First Natl. Bank Bldg., Jackson, Tenn., OALR
DANIELS, R. L.....Elk's Bldg., New Bern, N. C., OALR
DARMER, GEO. A.(1921)..401 Terminal Bldg., Aurora, Ill., OALR
*DARROW, C. H.(1928)
110 Metropolitan Bldg., Denver, Colo., ALR
DAVIDSON, H. P.(1924) 58 E. Washington St., Chicago, Ill., Op.
*DAVIES, J. S.(1927) 704 St. Helens Ave., Tacoma, Wash., OALR
DAVIES, T. A.(1913)....578 Sherborne St., Toronto, Can., OALR
DAVIS, A. EDWARD..40 E. 61st St., New York City, N. Y., Op.
*DAVIS, CHARLES L.(1928)
3542 Washington Ave., St. Louis, Mo., ALR
*DAVIS, F. A.(1927)
224 W. Washington Ave., Madison, Wis., OALR
DAVIS, G. E.....17 W. 74th St., New York City, N. Y., OALR
*DAVIS, HARRY B.(1920)
712 Bryant Bldg., Kansas City, Mo., OALR
*DAVIS, JOHN CALVIN JR.(1928)
425 Aquila Court, Omaha, Neb., OALR
*DAVIS, J. E.....Kress Bldg., McAlester, Okla., OALR
DAVIS, J. L.....135 S. 18th St., Philadelphia, Pa., ALR
*DAVIS, RALPH F.(1920).....Mayer Bldg., Portland, Ore., OALR
DAVIS, THOMAS W.,
210 O'Hanlon Bldg., Winston-Salem, N. C., OALR
*DAVIS, W. T.(1919).927 Farragut Sq., Washington, D. C., Op.
DAYTON, G. O.....245 Bradbury Bldg., Los Angeles, Cal., OALR
DEAL, JOHN FRANCIS(1928)
1st Natl. Bk. Bldg., Springfield, Ill., OALR
*DEAN, ALFRED
308 Medical Arts Bldg., Grand Rapids, Mich., Op.
DEAN, F. W.(1904)....532 First Ave., Council Bluffs, Ia., OALR
*DEAN, L. W.....Washington University, St. Louis, Mo., ALR
*DEAN, WALTER(1917)..300 Frances Bldg., Louisville, Ky., OALR
DEBOE, M. P.....1st Natl. Bk. Bldg., Miami, Fla., OALR
DECHERD, H. B.....1515 Med. Arts Bldg., Dallas, Tex., OALR

DECKER, J. C.(1918).....Francis Bldg., Sioux City, Ia., OALR
*DEFNET, WM. A.(1921)..1309 Stroh Bldg., Detroit, Mich., ALR
DEICHLER, LYN W...1737 Chestnut St., Philadelphia, Pa., OALR
DELPH, JOHN FRANCIS(1928)
104 S. Michigan Blvd., Chicago, Ill., ALR
DENMAN, IRA O.(1915).....425 Ohio Bldg., Toledo, O., OALR
DENNIS, DAVID N.(1902).....221 W. Ninth St., Erie, Pa., Op.
DENNIS, EDWARD P.(1921).....309 W. 10th St., Erie, Pa., OALR
*DENNIS, F. L.(1906)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
DENNIS, GEO. J.(1910).25 E. Washington St., Chicago, Ill., ALR
DERBY, GEO. S.....23 Bay State Rd., Boston, Mass., Op.
DETLING, FRANK E...500 S. Lucas St., Los Angeles, Cal., OALR
DERIVER, J. P.(1919)
Veterans Bureau, San Francisco, Cal., OALR
DEWEESE, CLARENCE...154 N. Upper St., Lexington, Ky., OALR
DEWEY, C. H.(1912)
Sternberg General Hospital, Manila, P. I.
DEWITT, EDWARD NICHOLAS(1928)
836 Myrtle Ave., Bridgeport, Conn., Op.A.
DICKINSON, B. M., 637 Union Trust Bldg., Pittsburgh, Pa., ALR
DICKSON, JOHN FOREST(1928)
418 Mayer Bldg., Portland, Ore., OALR
DINTENFASS, HENRY...1305 Spruce St., Philadelphia, Pa., ALR
DIXON, G. S.....218 2nd Ave., New York City, N. Y., R
*DIXON, OTTO JASON(1928)
902 Med. Arts Bldg., Kansas City, Mo., ALR
DIXON, W. E..503 Med. Arts Bldg., Oklahoma City, Okla., OALR
DOANE, L. L.(1905).....357 N. Main St., Butler, Pa., OALR
DODD, OSCAR(1903).....708 Church St., Evanston, Ill., Op.
DOHERTY, WM. B.(1927)
150 W. 55th St., New York City, N. Y., Op.
DOLMAN, PERCIVAL(1919)
490 Post St., San Francisco, Cal., Op.
*DONNELL, N. R.(1917) Metropolitan Bldg., St. Louis, Mo., Op.
*DONOHER, WM. D....Boston Bldg., Salt Lake City, Utah, OALR
DONOVAN, JNO. A.(1902) 507 Phoenix Bldg., Butte, Mont., OALR
DORENTE, D. R....1st Natl. Bank Bldg., Fort Smith, Ark., OALR
DORGE, RICHARD IRVING(1922)
Silvertsen Clinic, Minneapolis, Minn., OALR
DORSEY, J. G.....201 N. Main St., Wichita, Kan., OALR
DOUGLAS, FREDERICK ALEXANDER(1922)
303 State Bank Bldg., La Crosse, Wis., OALR
DOW, FRANK E.(1914)
4 West St., Northampton, Mass., Op.
DOWLING, J. IVIMEY,
116 Washington Ave., Albany, N. Y., OALR
DOWLING, J. T.(1915)
Virginia Mason Hosp. Bldg., Seattle, Wash., OALR

*Duane, J. F.(1916).....Jefferson Bldg., Peoria, Ill., OALR
Dulaney, Octavus.....811 Brown Bldg., Louisville, Ky., ALR
*Dunlap, Lawrence G.(1920)
Electric Light Bldg., Anaconda, Mont., OALR
Dunn, Murison.....Richmond, Ky., OALR
Dunn, O. B.....4th and Railroad Sts., Ironton, O., OALR
*Dunnington, J. H.(1924)
30 W. 59th St., New York City, N. Y., Op.
*Duntley, G. S.(1913)....103 So. Side Sq., Macomb, Ill., OALR
Durr, S. A.(1927)
1304 Medico-Dental Bldg., San Diego, Cal., Op.
Dutrow, H. V.(1914)....1040 Fidelity Bldg., Dayton, O., OALR
*Dyer, C. P.(1920)...408 Humboldt Bldg., St. Louis, Mo., Op.

E

*Earel, A. M.(1912).....857 Pleasant St., Hoopeston, Ill., OALR
Earley, James Hugh(1928)
1801 Eye St., N. W., Washington, D. C., ALR
Easton, J. C.(1904)
First Nat'l Bank Bldg., Springfield, O., OALR
Eber, Carl Theodore(1928)
1006 Carleton Bldg., St. Louis, Mo., Op.
Edson, R. A.....498 Delaware Ave., Buffalo, N. Y., Op.
*Edwards, C. J.(1913)
First National Bank Bldg., Vicksburg, Miss., OALR
Edwards, Slocomb R.(1918)
Christman Bldg., South Bend, Ind., OALR
*Egan, Burton Wright(1928)
408 North St., Logansport, Ind., OALR
Eimer, Charles Eugene(1928)
316 Lister Bldg., St. Louis, Mo., ALR
Ellegood, J. A.(1898)
408 Equitable Bldg., Wilmington, Del., OALR
*Ellett, E. C.(1898)....Exchange Bldg., Memphis, Tenn., Op.
*Ellis, Charles Ward(1928).....Lansing, Mich., ALR
Ellis, Z. H.(1927) 30 W. 59th St., New York City, N. Y., Op.
Elwood, Calvin R.(1922).Spies Bldg., Menominee, Mich., OALR
Embery, Frank(1927)
4660 Frankford Ave., Philadelphia, Pa., ALR
*Emerson, Linn(1906) Metropolitan Bldg., Orange, N. J., OALR
Engle, H. P.....Maytag Bldg., Newton, Ia., OALR
Ersner, Matthew S..1915 Spruce St., Philadelphia, Pa., ALR
Estabrook, Chas. T..857 Pleasant St., Worcester, Mass., OALR
*Eubank, Ambrose E.(1928)
636 Argyle Bldg., Kansas City, Mo., OALR
*Evans, J. N.(1925).23 Schermerhorn St., Brooklyn, N. Y., Op.
*Ewing, Arthur E....Metropolitan Bldg., St. Louis, Mo., Op.
*Ezell, H. C.(1926)..228 Doctors Bldg., Nashville, Tenn., OALR

F

FAIRBAIRN, JOHN F....503 Delaware Ave., Buffalo, N. Y., ALR
*FAIRCHILD, NORA M.(1922) Med. Arts Bldg., Omaha, Neb., Op.
FAIRING, JOHN W.(1924)
206 Tribune Review Bldg., Greensburg, Pa., OALR
FAITH, THOMAS.....31 N. State St., Chicago, Ill., OALR
*FALLOWS, HOWARD D.(1916)
Park Hospital Clinic, Mason City, Ia., OALR
*FARMER, A. G.(1919).....Fidelity Bldg., Dayton, O., OALR
FARNSLER, H. HERSHEY.1438 Market St., Harrisburg, Pa., OALR
FARNSWORTH, M. A.(1917)..2436-6th St., Boulder, Colo., OALR
*FARRELL, P. J. H.(1902)
25 E. Washington St., Chicago, Ill., OALR
FARRINGTON, P. M.(1904)
Physicians and Surgeons Bldg., Memphis, Tenn., ALR
*FARRIOR, J. BROWN(1928)
605 Citizens Bank Bldg., Tampa, Fla., OALR
*FAWCETT, I.(1913).....75 12th St., Wheeling, W. Va., OALR
FELLOWS, C. G.....30 N. Michigan Blvd., Chicago, Ill., OALR
FELT, C. L.....Chestnut & 20th Sts., Philadelphia, Pa., ALR
*FENTON, RALPH A.(1921)
806 Medical Arts Bldg., Portland, Ore., OALR
*FERGUSON, E. S.(1906)
902 Medical Arts Bldg., Oklahoma City, Okla., OALR
FERGUSON, R. J.(1927)
59 College St., New Haven, Conn., OALR
FERMAN, JOHN W.(1921).Stahlman Bldg., Charleroi, Pa., OALR
*FERRIS, DAVID P.(1928)
1000 Carleton Bldg., St. Louis, Mo., ALR
FERNLUND, GEORGE BYRON(1928)
Kew Arms, Kew Gardens, New York, OALR
FIELDING, F. G.(1925)....70 Ridge St., Glens Falls, N. Y., ALR
FIGI, F. A.(1927).....Mayo Clinic, Rochester, Minn., LR
FINK, WALTER H.(1927)
426 LaSalle Bldg., Minneapolis, Minn., OALR
*FINNOFF, W. C.(1916)..217 Imperial Bldg., Denver, Colo., Op.
FISCHER, N. A.(1925)
623 Union Trust Bldg., Pittsburgh, Pa., ALR
FISCHER, V. B...230 Mercantile Bk. Bldg., Boulder, Colo., OALR
FISHER, CARL.....1930 Wilshire Blvd., Los Angeles, Cal., Op.
FISHER, JAMES A.(1925).Fitkin Bldg., Asbury Park, N. J., OALR
*FISHER, W. A.(1896)
231 W. Washington St., Chicago, Ill., OALR
FISKE, GEO. F.....25 E. Washington St., Chicago, Ill., OALR
*FITZGERALD, J. P.(1924)
104 S. Michigan Blvd., Chicago, Ill., Op.
FLAGG, J. D.(1900).....473 Virginia St., Buffalo, N. Y., Op.A
FLATLEY, T. J.(1914).....Reliance Bldg., Moline, Ill., OALR
FLOYD, T. W.(1918)...811 Peoria Life Bldg., Peoria, Ill., OALR

FLYNN, CHAS. T.(1927)
1138 Chapel St., New Haven, Conn., ALR
FLYNN, JAMES A.(1924)
1511 Rhode Island Ave., N. W., Washington, D. C., OALR
*FOGARTY, CHAS. W.(1920)
1118 Lowry Bldg., St. Paul, Minn., OALR
FOLEY, J. W.(1915).....Majestic Bldg., Denver, Colo., OALR
FOLK, MAX LYON(1928)
55 E. Washington St., Chicago, Ill., OALR
*FOLKEN, FRANK GARRETT(1928)
306 Albert Lea State Bank Bldg., Albert Lea, Minn., OALR
*FOLSOM, W. H.(1927).11 N. Main St., Fond du Lac, Wis., OALR
FORBES, SHERMAN B.(1921)
409 Citizens Bank Bldg., Tampa, Fla., OALR
*FORD, HANBY LEWIS(1928)
401 Robeson Bldg., Champaign, Ill., OALR
*FORGRAVE, L. ROBERT(1922)
204 Logan Bldg., St. Joseph, Mo., OALR
FORSYTHE, S. T.(1919) 235 Colonial Arcade, Cleveland, O., OALR
FORT, A. G.....303 Medical Arts Bldg., Atlanta, Ga., OALR
FOSTER, J. H.....1306 Walker Ave., Houston, Tex., ALR
*FOUTS, JOHN D.(1920)....910 Fidelity Bldg., Dayton, O., OALR
*FOWLER, EARL B.(1925) 55 E. Washington St., Chicago, Ill., Op.
FOWLER, S. R.....University Bldg., Syracuse, N. Y., OALR
*FOWLER, WM.(1925).1048 Maccabee Bldg., Detroit, Mich., OALR
*FOX, C. H.(1920).....2119 Central Ave., Kearney, Neb., OALR
FOX, L. WEBSTER.....303 S. 17th St., Philadelphia, Pa., Op.
FOX, NOAH(1926)..6306 Cottage Grove Ave., Chicago, Ill., ALR
FOX, RALPH D.(1916).....Unity Bldg., Bloomington, Ill., OALR
FRANCHERE, FRED'K E.(1920)
Toy Bank Bldg., Sioux City, Ia., OALR
FRANK, IRA(1908)....104 S. Michigan Blvd., Chicago, Ill., ALR
FRANKEL, EDWARD, JR.(1928)
152 W. 58th St., New York City, N. Y., ALR
FRASER, R. H.(1925)
Battle Creek Sanitarium, Battle Creek, Mich., ALR
FRASER, W. G.(1927).....52 Nepean St., Ottawa, Canada, OALR
FREEDMAN, L. M.(1920)
475 Commonwealth Ave., Boston, Mass., ALR
*FREIMUTH, L. E.(1927)
234 University Club Bldg., St. Louis, Mo., ALR
*FRENCH, R. F.(1913).Masonic Temple, Marshalltown, Ia., OALR
FREUDENTHAL, WOLFF,
24 W. 88th St., New York City, N. Y., ALR
FREY, CLARENCE L.....Dime Bank Bldg., Scranton, Pa., OALR
FRIEDENWALD, HARRY....1212 Eutaw Pl., Baltimore, Md., Op.A
*FRIEDENWALD, J. S.(1925) 1212 Eutaw Pl., Baltimore, Md., Op.
FRINGER, W. R.....William Brown Bldg., Rockford, Ill., Op.
*FROST, ALBERT DALBEY(1928)
681 E. Broad St., Columbus, O., Op.

FROTHINGHAM, EDWARD (1928)
8644 94th St., Woodhaven, N. Y., OALR
*FUCHS, VALENTINE HENRY (1928)
220 Chaille Bldg., New Orleans, La., ALR
FULKERSON, C. B. (1914)
308 Kalamazoo Nat'l Bank Bldg., Kalamazoo, Mich., OALR
FULLENWIDER, C. M. . 404 Barnes Bldg., Muskogee, Okla., OALR
FULLER, JOHN ANDRE (1928) . . . . . Reno, Nev., OALR
FULLER, HARRY B. (1925) . . . 2219 E. 70th Pl., Chicago, Ill., OALR
*FULLER, T. E. (1915)
Texarkana Natl. Bank Bldg., Texarkana, Tex., OALR
FULTON, JOHN F. (1920) 833 Lowry Bldg., St. Paul, Minn., OALR
FURGASON, A. P. . . . . Richards Blk., Lincoln, Neb., OALR
FURSTENBERG, A. C. (1925)
330 S. State St., Ann Arbor, Mich., ALR
FUST, JOHN H. E. (1922) . . . . . 154 W. 8th St., Erie, Pa., OALR

G

*GADBOIS, ARTHUR E. (1921)
421 Norfolk Ave., Norfolk, Neb., OALR
*GAILEY, WATSON W. (1923)
Griesheim Bldg., Bloomington, Ill., OALR
GALE, M. JEAN (1919) . . . . . Republic Bldg., Denver, Colo., Op.
GALLAGHER, J. H. C. (1927)
1537 Main St., Springfield, Mass., ALR
GALLAHER, T. J. . . . . California Bldg., Denver, Colo., ALR
*GALLOWAY, T. C. (1927) . . . . 636 Church St., Evanston, Ill., ALR
GARBER, J. M. . . . . 14 N. Mulberry St., Mansfield, O., OALR
*GARRAGHAN, EDW. F. . 25 E. Washington St., Chicago, Ill., OALR
GARRETT, J. R. (1913) . . . . . Mt. Trust Bldg., Roanoke, Va., OALR
GATEWOOD, E. T. (1925) Professional Bldg., Richmond, Va., ALR
GATEWOOD, W. L. . . . 330 Park Ave., New York City, N. Y., ALR
*GAUDET, LUCIEN SYDNEY (1928)
405 Franklin St., Natchez, Miss., OALR
GAVIN, EDWARD F. . . . . 1 S. Sheridan Rd., Waukegan, Ill., OALR
*GEIGER, C. W. (1904) . . . . . Cobb Bldg., Kankakee, Ill., OALR
*GEORGE, HENRY W. (1919)
19 N. Union St., Middletown, Pa., Op.
GIBSON, R. D. (1907) . . . . 310 Wick Ave., Youngstown, O., OALR
*GIFFORD, HAROLD. . . . . Med. Arts Bldg., Omaha, Neb., Op.
*GIFFORD, S. R. (1919) . . . . . Med. Arts Bldg., Omaha, Neb., Op.
GILBERT, FRANK Y. . . . . 148 Park St., Portland, Me., OALR
*GILL, ARTHUR ERNEST (1928)
U. S. Veterans' Hosp., No. 88, Memphis, Tenn., OALR
GILL, E. G. (1919) . . . . . 711 S. Jefferson St., Roanoke, Va., ALR
*GILL, G. BACHE (1923)
1624 Eye St., N. W., Washington, D. C., ALR
GILL, GEORGE (1917)
Elyria Savings & Trust Bldg., Elyria, O., OALR
*GILLETT, WILBUR G. . . . . 201 N. Main St., Wichita, Kas., Op.

GILLETTE, D. F.(1925)...300 Univ. Blk., Syracuse, N. Y., Op.
*GILLUM, J. R.(1920)...221 S. 6th St., Terre Haute, Ind., OALR
GIPNER, J. F.(1926)....53 Fitzhugh St., Rochester, N. Y., Op.
GITTENS, T. R.(1920).401 Davidson Bldg., Sioux City, Ia., OALR
GLASFORD, S. T.(1924).....Pekin, Ill., OALR
GLEASON, E. B.....2112 Pine St., Philadelphia, Pa., ALR
*GLICK, HARRY NAYLOR(1928)
3115 S. Grand Ave., St. Louis, Mo., ALR
GLOGAU, O.,
1130 Park Ave., cor. 91st St., New York City, N. Y.. ALR
*GLOSSER, HERBERT H.(1912)
448 Franklin St., Buffalo, N. Y., Op.
GLOVER, S. P.....1118 Twelfth Ave., Altoona, Pa., OALR
GLOWACKI, BEN FRANCIS(1928)
770 Maccabee Bldg., Detroit, Mich., OALR
*GOAR, EVERETT LOGAN(1926)
1300 Walker Ave., Houston, Tex., Op.
GODDARD, H. M.....1531 Spruce St., Philadelphia, Pa., ALR
GOLDBACK, L. J.(1911)
1012 N. Charles St., Baltimore, Md., Op.
GOLDING, JOSEPH E.(1925)
170 Hancock St., Brooklyn, N. Y., Op.
GOLDMAN, A. MILTON(1928)
121 E. 60th St., New York City, N. Y., Op.
GOLDMAN, H. G....57 W. 58th St., New York City, N. Y., ALR
*GOLDSMITH, P. G.....84 Carlton St., Toronto, Can., ALR
*GOLDSTEIN, M. A.(1896)
3858 Westminster Pl., St. Louis, Mo., ALR
*GOODYEAR, H. M.(1924) 556 Doctors Bldg., Cincinnati, O., ALR
GORDON, CHAS. H.(1926).....46 Deering St., Portland, Me., ALR
GORDON, J. S.(1919)
141 E. Wisconsin St., Milwaukee, Wis., OALR
GORRELL, T. J. H.,
16th & Vincennes Ave., Chicago Heights, Ill., OALR
*GOSNEY, C. W.(1920).1009 Haas Bldg., Los Angeles, Cal., Op.
*GOTTLIEB, MARK J.23 W. 73d St., New York City, N. Y., ALR
*GOURLY, G. F.(1916).....Sinclair Bldg., Steubenville, O., OALR
*GOUX, RAYMOND SALOT,
545 David Whitney Bldg., Detroit, Mich., OALR
*GRACEY, GEO. F.....209 State St., Harrisburg, Pa., OALR
*GRADLE, H. S.(1911)..58 E. Washington St., Chicago, Ill., Op.
GRAHAM, H. B.....490 Post St., San Francisco, Cal., ALR
GRANGER, W. B.(1920)
Citizens Natl. Bank Bldg., Emporia, Kan., OALR
*GRANT, FRED. E.(1912)
Kal. Natl. Bank Bldg., Kalamazoo, Mich., OALR
*GRANT, LEE E.(1919) David Whitney Bldg., Detroit, Mich., OALR
GRANT, P. T.(1919)
1407 Grand Rapids Natl. Bk. Bldg., Grand Rapids, Mich., OALR
GRAY, E. B.(1927)....Mercer Bldg., New London, Conn., ALR
*GREEN, A. S..The Green's Eye Hosp., San Francisco, Cal., Op.

*GREEN, JOHN (1905)
3720 Washington Blvd., St. Louis, Mo., Op.
GREEN, L. D. . The Green's Eye Hosp., San Francisco, Cal., Op.
*GREENE, JOSEPH BERRY (1923)
305 Haywood Bldg., Asheville, N. C., ALR
*GREENE, LAURENCE WHITRIDGE (1928)
806 Metropolitan Bldg., Denver, Colo., ALR
GREENFIELD, WILLIAM JOHN (1928)
The Polyclinic, 20 S. Dunlap St., Memphis, Tenn., ALR
GREENFIELD, SAMUEL D. (1927)
169 New York Ave., Brooklyn, N. Y., ALR
*GREENWOOD, A. (1911)
82 Commonwealth Ave., Boston, Mass., Op.
*GREEVER, B. L. (1920)
610 Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
GREGG, JOHN B. . . 231 N. Summit Ave., Sioux Falls, S. D., OALR
GRIM, U. J. . . . . 30 N. Michigan Blvd., Chicago, Ill., OALR
GRISCOM, J. MILTON. . . 2213 Walnut St., Philadelphia, Pa., Op.
GROSS, JULIUS E. (1920)
8030 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
GROSVENOR, L. N. (1907)
366 Dakota Ave., So. Huron, S. D., OALR
*GROVE, W. E. (1914)
120 E. Wisconsin Ave., Milwaukee, Wis., OALR
GRUSHLOW, ISRAEL,
271 Central Pk., W., New York City, N. Y., ALR
*GSELL, J. F. (1920) . . . . . 911 Beacon Bldg., Wichita, Kan., OALR
GUERINOT, A. J. (1918)
604 Westinghouse Bldg., Pittsburgh, Pa., OALR
GUGGENHEIM, L. K. . . . . Carleton Bldg., St. Louis, Mo., ALR
GUNDELACH, C. A.,
825 University Club Bldg., St. Louis, Mo., ALR
GUTHRIE, A. L. (1910)
1010 Medical Arts Bldg., Oklahoma City, Okla., OALR
GUTTMAN, J. . . . 987 Madison Ave., New York City, N. Y., OALR
*GUYTON, B. S. (1918) . . . . . Oxford, Miss., OALR
GWINN, G. E. (1916)
704 Central Trust Bldg., San Antonio, Tex., OALR

H

*HAAS, FREDERICK J. (1920)
Masonic Bldg., Leavenworth, Kan., OALR
*HACKNEY, F. J. (1913)
630 Volunteer Bldg., Chattanooga, Tenn., OALR
HADEN, HENRY C. . . . Niels-Esperon Bldg., Houston, Tex., Op.
HAGEMANN, JOHN A. (1925)
Highland Bldg., Pittsburgh, Pa., ALR
HAGLER, E. E. (1896) . . . . . Hagler Bldg., Springfield, Ill., OALR

HALE, ARTHUR STANLEY (1928)
3790 Woodward Ave., Detroit, Mich., OALR
*HALL, ALICE K. (1924). 25 E. Washington St., Chicago, Ill., ALR
*HALL, EDWIN PERRY (1928)
State Tower Bldg., Syracuse, N. Y., ALR
HALL, G. C. (1907). . . . . 705 Brown Bldg., Louisville, Ky., OALR
HALLETT, G. DEWAYNE (1927)
274 W. 86th St., New York City, N. Y., Op.
HALSTED, FRED S. . . 728 Metropolitan Bldg., Denver, Colo., OALR
*HAMLIN, FRED EUGENE (1926)
Shenandoah Life Bldg., Roanoke, Va., ALR
HAMPTON, ROBT. R.,
804 Boston Bldg., Salt Lake City, Utah, ALR
HANCOCK, J. C. (1914)
135 Cambridge Pl., Brooklyn, N. Y., Op.
*HANDS, SIDNEY GILLOTT (1926)
602 1st Nat'l Bank Bldg., Davenport, Ia., ALR
HANEY, WM. P. (1920)
1500 Medical Arts Bldg., Omaha, Neb., OALR
HANS, C. L. (1921). . . . . 19 W. 7th St., Cincinnati, O., OALR
*HANSEL, FRENCH KELLER (1926)
Beaumont Bldg., St. Louis, Mo., ALR
*HANSEN, ERLING W. (1920)
910 Donaldson Bldg., Minneapolis, Minn., OALR
*HARBERT JOHN P. (1918)
135 N. Main St., Bellefontaine, O., OALR
*HARDESTY, J. F. (1920)
1021 Missouri Bldg., St. Louis, Mo., Op.
HARDING, FREDERICK B. (1926)
941 Hamilton St. (Medical Arts Bldg.), Allentown, Pa., OALR
HARDY, LE GRAND HAVEN (1928)
30 E. 40th St., New York City, N. Y., Op.
*HARDY, W. F. (1914). . . . . Humboldt Bldg., St. Louis, Mo., Op.
*HARGITT, CHARLES A. (1926)
17 Schermerhorn St., Brooklyn, N. Y., Op.
*HARKNESS, GORDON F. (1911)
Putnam Bldg., Davenport, Ia., OALR
*HARRELL, VOSS (1926). . . . . Hofman Bldg., Detroit, Mich., ALR
HARRIS, C. M. (1910)
604 Johnstown Trust Bldg., Johnstown, Pa. OALR
HARRIS, H. B. . . . . Fidelity Med. Bldg., Dayton, O., OALR
HARRIS, W. C. (1900). . . . 7th and Race St., Cincinnati, O., ALR
HARRIS, W. ROBIN (1923)
Physicians and Surgeons Bldg., Memphis, Tenn., ALR
HARRISON, FRED HAZARD (1928)
308 First Natl. Bank Bldg., Connellsville, Pa., OALR
HARRISON, W. G. . . . . Empire Bldg., Birmingham, Ala., OALR
HARTLEY, F. A. . . . . 302 E. High St., Springfield, O., OALR
HARTSHORNE, ISAAC (1926)
30 W. 59th St., New York City, N. Y., Op.

HARTZEL, S. M.....Dollar Bank Bldg., Youngstown, O., OALR
HARVEY, N. D.....112 Waterman Ave., Providence, R. I., OALR
HASELTINE, BURTON(1927)
122 S. Michigan Ave., Chicago, Ill., OALR
HASKELL, ALFRED W.....129 Park St., Portland, Me., Op.
HASKIN, W. H.(1913)..57 W. 57th St., New York, N. Y., ALR
HASPEL, M. DAVID(1911)
603 Maison Blanche Bldg., New Orleans, La., OALR
*HASTY, F. E.(1925) Medical Arts Bldg., Nashville, Tenn., ALR
HATCH, C. B.(1908).....3 W. Church St., Newark, O., OALR
HAUER, A. M.(1916).....327 E. State St., Columbus, O., ALR
HAUGHEY, WILFRID(1911)
303 Post Bldg., Battle Creek, Mich., OALR
HAWLEY, C. W.....Med. Arts Bldg., Chicago, Ill., OALR
HAWMAN, ERLE G...4051 N. Broad St., Philadelphia, Pa., Op.
*HAYDEN, AUSTIN A.(1910)
25 E. Washington St., Chicago, Ill., OALR
HAYS, HAROLD M.(1912)
22 W. 74th St., New York City,N. Y., ALR
HAZLETT, L. R.(1913).Savings & Trust Bldg., Butler, Pa., OALR
HEALEY, JOHN F.(1924)503 Delaware Ave., Buffalo, N. Y., ALR
HEARD, C. F.(1913).....813 Sassafrass St., Erie, Pa., OALR
HEARD, MARY K.....41 W. Main St., North East, Pa., Op.
*HEATH, PARKER(1924)
David Whitney Bldg., Detroit, Mich., Op.
HEBERT, ARTHUR W.....Flood Bldg., San Francisco, Cal.,
HECKEL, EDWARD B.(1902)
Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
HEEB, HARRY J....204 Wisconsin Ave., Milwaukee, Wis., OALR
HEED, C. R.....1205 Spruce St., Philadelphia, Pa., Op.
HEGGIE, N. M.(1905)...Buckman Bldg., Jacksonville, Fla., OALR
*HEITGER, J. D.(1905)...808 Francis Bldg., Louisville, Ky., OALR
HEMPSTEAD, BERT E.....Mayo Clinic, Rochester, Minn., ALR
HENNESSEY, WM. W.....333 Essex St., Salem, Mass., OALR
HENNING, CARL..1610 Eye St., N. W., Washington, D. C., Op.
HENNINGER, L. L.(1913)
205 Citizens Bank Bldg., Pasadena, Cal., OALR
HENTON, G. E.(1918).....Morgan Bldg., Portland, Ore., OALR
HERRIMAN, L. L.(1913).....Alamosa, Colo., OALR
HESS, WM. L.(1904)...1554 California St., Denver, Colo., OALR
HETRICK, L. E.(1923)
40 E. 61st St., New York City, N. Y., ALR
HETZEL, C. C.(1906).....Eccles Bldg., Ogden, Utah, OALR
HICKS, VONNIE MONROE(1926)
Professional Bldg., Raleigh, N. C., Op.
*HIGBEE, D. R.(1927)....3245 Fourth St., San Diego, Cal., ALR
HIGGINS, R. P.(1924).....20 Court St., Cortland, N. Y., OALR
*HIGGINS, S. G...120 E. Wisconsin Ave., Milwaukee, Wis., OALR
HILL, C. E.(1919).....160 Bloor St., W., Toronto, Can., Op.
*HILL, EMORY(1916)...Profesional Bldg., Richmond, Va., Op.

*Hill, Frederick Thayer (1928)
Professional Bldg., Waterville, Me., ALR
Hill, Howard Foster (1928)
Professional Bldg., Waterville, Me., Op.
Hinman, E. E. (1924) 146 Washington Ave., Albany, N.Y., ALR
Hitz, H. B. . . . . 141 E. Wisconsin Ave., Milwaukee, Wis., ALR
Hodson, B. F. . . . . 418 Security Bldg., Miami, Fla., OALR
*Hoffman, John N. (1927) . . . 308 Brant Bldg., Canton, O., ALR
Hoffman, L. G. (1927) 30 N. Michigan Ave., Chicago, Ill., OALR
*Hofman, Walter F. (1920) . 817 Summit, Seattle, Wash., Op.
*Hofmann, W. P. (1925) . . 707 Kahl Bldg., Davenport, Ia., OALR
Hogan, W. L. (1924) . . . . . 750 Main St., Hartford, Conn., OALR
*Hogshead, J. M. . . . . Hogshead Apt., Chattanooga, Tenn., OALR
Hogue, D. W. (1908) 1007 Tecumseh Bldg., Springfield, O., OALR
Hohf, Julius (1915) . . . . . Clinic Bldg., Yankton, S. D., OALR
Holdsworth, Frank
People's Bank Bldg., Traverse City, Mich., OALR
Holloway, Thos. B. . 1819 Chestnut St., Philadelphia, Pa., Op.
Holmes, Benjamin H. . . . . 435 Baker Blk., Racine, Wis., OALR
Holt, E. E., Jr. . . . . 723 Congress St., Portland, Me., Op.
*Hompes,, J. J. . . . . Security Mutual Bldg., Lincoln, Neb., OALR
Hood, T. C. (1904) Hume-Mansur Bldg., Indianapolis, Ind., Op.
Hopkins, W. E. . . . . 515 Park Ave., New York City, N.Y., OALR
*Hourn, George E. (1920)
511 University Club Bldg., St. Louis, Mo., ALR
Hovde, A. G. (1916) Board of Trade Bldg., Superior, Wis., OALR
Howard, C. N. (1919) . . . 210 S Indiana St., Warsaw, Ind., OALR
Howard, E. F. (1919)
First Natl. Bank Bldg., Vicksburg, Miss., ALR
Howard, Edward L. (1926)
405 Medical Arts Bldg., Fort Worth, Tex., OALR
*Howard, Francis C. (1928)
909 Univ. Club Bldg., St. Louis, Mo., ALR
*Howard, Harvey (1928)
School of Med., Washington University, St. Louis, Mo., Op.
Howard, J. W. . . . . Rialto Bldg., Kansas City, Mo., OALR
Howard, Lloyd G. (1923)
Council Bluffs Clinic, Council Bluffs, Iowa, OALR
Howard, W. H. (1923)
1009 Nicollet Ave., Minneapolis, Minn., OALR
*Howe, A. W. (1922)
1616 Washington Bldg., Tacoma, Wash., OALR
Hughes, J. A. (1927) . . . Exchange Bldg., Memphis, Tenn., OALR
Hughes, L. W. (1925) . . . . 1019 Broad St., Newark, N. J., OALR
Hughes, Ray Whitbeck (1928)
1501 David Whitney Bldg., Detroit, Mich., OALR
Hughes, T. E. (1922) . . 1000 W. Grace St., Richmond, Va., ALR
*Hughes, W. F.,
401 Hume-Mansur Bldg., Indianapolis, Ind., Op.
Hurd, L. M. (1910) 39 E. 50th St., New York City, N. Y., ALR
Hurff, J. W. (1919) . . . 86 Washington St., Newark, N. J., OALR

HURLEY, E. D.(1914)....419 Boylston St., Boston, Mass., Op.
*HURST, V. REEVES(1923)
107 E. Methvin St., Longview, Tex., OALR
HUSIK, D. N.(1921)....1610 Spruce St., Philadelphia, Pa., ALR
HUSSEY, E. J.....245 Maple St., Holyoke, Mass., OALR
HUTCHINSON, ABBOTT TRASK(1928)
30 W. 59th St., New York City, N. Y., ALR

I

*IGLAUER, S.....707 Race St., Cincinnati, O., ALR
IMPERATORI, C. J....18 E. 38th St., New York City, N. Y., ALR
INGERMAN, S. M.(1909)
418 Central Park, W., New York City, N. Y., OALR
INGERSOLL, J. M.(1908)
1700 S. Bayshore Lane, Miami, Fla., ALR
INGLIS, HARRY J.....43 Bay State Rd., Boston, Mass., ALR
*INGRAM, L. C.(1915).....Clinic Bldg., Orlando, Fla., OALR
IRVIN, EDGAR H..903 First Natl. Bk. Bldg., El Paso, Tex., OALR
IRVINE, A. R.(1918)
1144 Roosevelt Bldg., Los Angeles, Cal., Op.
IRWIN, FRANK N.(1905)
114 E. 54th St., New York City, N. Y., Op.
IRWIN, V. J.....Riverview, Fla., OALR
IRWIN, VINCENT J., JR.(1921)
1634 Main St., Springfield, Mass., OALR
ISRAEL, NORMA E.(1919)
2001 Niels Esperon Bldg., Houston, Tex., OALR
ISRAEL, SIDNEY..2001 Niels Esperon Bldg., Houston, Tex., ALR
IVINS, H. M.(1910)
815 Merchants Natl. Bank Bldg., Cedar Rapids, Ia., OALR

J

JACKLE, ARTHUR F.(1917)
63 E. Fourth St., Dunkirk, N. Y., Op.
*JACKSON, ATRAS EARL(1928)
Med. Arts Bldg., Fort Worth, Tex., Op.
JACKSON, CHEVALIER....235 S. 15th St., Philadelphia, Pa., L
JACKSON, J. D.(1920).....232 West 8th St., Erie, Pa., OALR
*JACOBS, MAX W.(1916)
Missouri Theatre Bldg., St. Louis, Mo., Op.
JACOBS, S. M....1018 E. 163rd St., New York City, N. Y., OALR
JACOBY, D. P. A.(1912).....106 Turo St., Newport, R. I., Op.AL
*JACQUISH, CHARLES JOSEPH(1928)
904 Westinghouse Bldg., Pittsburgh, Pa., ALR
JAMES, J. H.....Natl. City Bank Bldg., Mankato, Minn., OALR
JARECKY, HERMAN 168 W. 86th St., New York City, N. Y., OALR
JEAN, GEO. W.....7 East Valerio, Santa Barbara, Cal., Op.
*JENNINGS, J. ELLIS....807 Carleton Bldg., St. Louis, Mo., Op.

*Jessaman, L. W.(1914)
132 Union Ave., Framingham, Mass., OALR
*Jobson, G.B.(1903).....Printz Bldg., Franklin, Pa., OALR
Johnson, A. E.(1919).....Watertown, S. D., OALR
Johnson, H. R.(1913)..American Bldg., Fairmont, W. Va., OALR
Johnson, Otis H.....Morehead City, N. C., OALR
Johnson Thomas Holland(1923)
30 W. 59th St., New York City, N. Y., Op.
*Johnston, W. H.(1912).508 Laurel Bldg., Muscatine, Ia., OALR
*Johnston, Wilson(1917) 611 Lovejoy St., Portland, Ore., OALR
Jones, C. C.(1924)....656 Doctors' Bldg., Cincinnati, O., ALR
*Jones, Cecil Cain(1928)
1120 Equitable Bldg., Des Moines, Ia., OALR
Jones, E. L.(1913)
First Natl. Bank Bldg., Cumberland, Md., OALR
*Jones, Edley Hicks(1928)
First Natl. Bank Bldg., Vicksburg, Miss., OALR
Jones, Edmund Lloyd(1926)
Wheeling Clinic, Wheeling, W. Va., OALR
Jones, E. W.....44 Atlanta St., Lynn, Mass., Op.A
*Jones, F. W.(1919).....Times Bldg., Alton, Ill., OALR
Jones, L. W.(1925)..53 S. Fitzhugh St., Rochester, N. Y., OALR
Jones, U. G.(1927)
Jones Eye, Ear, Nose & Throat Hos., Johnson City, Tenn., ALR
Jones, Vincent L.(1928)
3511 Washington Ave., St. Louis, Mo., Op.
Jones, Wm. S.(1927)
522 Sheridan Rd., Menominee, Mich., OALR
Jordan, George T.(1921)
30 N. Michigan Blvd., Chicago, Ill., OALR
Joy, Harold Henry(1926)
504 State Tower Bldg., Syracuse, N. Y., Op.
*Joynt, A. J.(1924).....Black Bldg., Waterloo, Ia., OALR
Judd, H. B.(1917)..47 E. 57th St., New York City, N. Y., ALR
Judge, Harry V.(1920).....46 Willett St., Albany, N. Y., Op.

K

Kahn, M. E.(1925)..104 S. Michigan Ave., Chicago, Ill., ALR
*Karkeet, Robert Budd(1928)
1203 Selling Bldg., Portland, Ore., OALR
Kauffman, A. B.(1922)..Gurney Bldg., Syracuse, N. Y., ALR
Kaufman, A. S.(1917).1923 Spruce St., Philadelphia, Pa., ALR
*Kearby, D. O.(1917)
709 Hume-Mansur Bldg., Indianapolis, Ind., ALR
Keating, T. F.(1927)
5050 Woodward Ave., Detroit, Mich., OALR
Keeler, J. C.....Med. Arts Bldg., Philadelphia, Pa., ALR
Keim, W. F.(1924).....25 Roseville Ave., Newark, N. J., ALR
Keith, D. M.....Wm. Brown Bldg., Rockford, Ill., ALR

*KEITH, W. E.....Waldheim Bldg., Kansas City, Mo., OALR
KELLER, S. A.....Sioux Falls Clinic, Sioux Falls, S. D., OALR
*KELLEY, ISAAC D., JR.(1928)
811 Beaumont Med. Bldg., St. Louis, Mo., ALR
KELLY, JOSEPH DOMINIC(1928)
114 E. 54th St., New York City, N. Y., OALR
KEMLER, JOS.(1922).....1908 Eutaw Pl., Baltimore, Md., OALR
KENDALL, M. R.(1919).....Osborn Bldg., Cleveland, O., ALR
KENNEDY, ELDRED WESTON(1928)
707 Gas & Elect. Bldg., Rochester, N. Y., Op.
KENNEY, WM. L....Corby Forsee Bldg., St. Joseph, Mo., OALR
KENNON, W. G.(1924)...Doctors' Bldg., Nashville, Tenn., OALR
*KETTELKAMP, FRED O.(1926)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
KEY, BEN WITT(1914)
100 W. 59th St., New York City, N. Y., Op.
KEY, SAM N.....320 Littlefield Bldg., Austin, Tex., OALR
*KEYS, MORTON J.(1921)
100 Jones Blk., Victoria, B. C., Canada, OALR
KIEFER, HUGO A.(1915)
Brockman Bldg., Los Angeles, Cal., OALR
KIEHLE, F. A.....410 Med. Arts Bldg., Portland, Ore., OALR
*KILBOURNE, P. H.(1924).Fidelity Med. Bldg., Dayton, O., OALR
*KILLEEN, JOHN JOSEPH(1928)
104 S. Michigan Ave., Chicago, Ill., ALR
*KINCAID, J. H.....421 W. Church Ave., Knoxville, Tenn., OALR
KINCANNON, LEROY TAYLOR(1928)
The Woodward, Birmingham, Ala., OALR
*KING, CLARENCE(1910)
Union Central Bldg., Cincinnati, O., Op.
KING, CHAS. R.(1920).....471 Spitzer Bldg., Toledo, O., OALR
KING, ED.....707 Race St., Cincinnati, O., ALR
*KING, G. L.....537 E. Market St., Alliance, O., OALR
*KING, J. J.(1916)...30 E. 40th St., New York City, N. Y., ALR
KING, O. H.(1916) Dugan-Stuart Bldg., Hot Springs, Ark., OALR
*KINNE, SANFORD HALSEY(1928)
73 Main St., Binghamton, N. Y., ALR
*KIRBY, D. B.(1924) 30 W. 59th St., New York City, N. Y., Op.
KIRKPATRICK, SAMUEL....902 Alabama Ave., Selma, Ala., OALR
KISER, J. D...Central Natl. Bank Bldg., Bartlesville, Okla., OALR
KISTLER, O. F.....43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
*KISTNER, F. B.....471 Montgomery Drive, Portland, Ore., OALR
KITTLESON, THEODORE(1915).....Fergus Falls, Minn., OALR
*KLINE, H. G.(1912)..State Tower Bldg., Syracuse, N. Y., ALR
KLINEDINST, JOHN FERD.(1906)
220 S. George St., York, Pa., Op.A
KLINGER, THOS. O.(1920)
318 Landers Bldg., Springfield, Mo., OALR
*KNAPP, ARNOLD....10 E. 54th St., New York City, N. Y., Op.
*KNAPP, B.....Cleveland Life Bldg., Evansville, Ind., OALR
KNAPP, F. N.(1925)....329 Bradley Bldg., Duluth, Minn., OALR

*KNEEDLER, G. C.(1912) Professional Bldg., Pittsburgh, Pa., ALR
*KNODE, A. R.(1916)...708 Med. Arts Bldg., Omaha, Neb., ALR
*KOENIG, IVAN J.(1928).....40 North St., Buffalo, N. Y., Op.
KOLLOCK, C. W.....86 Wentworth St., Charleston, S. C., OALR
KOOBS, H. J.G.(1920) 117½ W. Walnut St., Rogers, Ark., OALR
*KOPETZKY, S. J...51 W. 73rd St., New York City, N. Y., ALR
KRAFT, OSCAR H...1518 N. Dearborn Pkwy., Chicago, Ill., Op.
KRAUSS, FREDERICK..1703 Chestnut St., Philadelphia, Pa., OALR
KREBS, ADOLPH(1914)....Jenkins Arcade, Pittsburgh, Pa., Op.
KRESS, GEO. H.....245 Bradbury Bldg., Los Angeles, Cal., OALR
KRIEGER, G. M...701 Washington St., Michigan City, Ind., OALR
*KRIEGER, WM. A.(1919)
35 Market St., Poughkeepsie, N. Y., OALR
KRIMSKY, JOSEPH(1928)
263 Eastern Parkway, Brooklyn, N. Y., OALR
KRUG, ERNEST F.(1908)
12 W. 44th St., New York City, N. Y., Op.
KRUSKAL, I. D.(1925)..863 Prospect Pl., Brooklyn, N. Y., Op.
*KUHN, HUGH A.(1928)..1st Trust Bldg., Hammond, Ind., ALR
KULLY, B. M.(1922).....Med. Arts Bldg., Omaha, Neb., ALR

L

LAESSLE, HENRY A.(1922)
The Lenox, 13th & Spruce Sts., Philadelphia, Pa., ALR
LA FORCE, B. D.....1140 Beverley Rd., Altadena, Cal., OALR
LA FORCE, E. F.(1908)
Iowa State Bk. Bldg., Burlington, Ia., OALR
*LAMB, HARVEY(1920)
826 Metropolitan Bldg., St. Louis, Mo., Op.
LAMB, ROBT. SCOTT....Stoneleigh Ct., Washington, D. C., Op.
*LANCASTER, W. B.(1913)
520 Commonwealth Ave., Boston, Mass., Op.
LANDES, GEORGE A.....Parsons, Kas., OALR
LANDFRIED, C. J....607 Baronne Bldg., New Orleans, La., ALR
LANING, J. H.(1918)..907 Rialto Bldg., Kansas City, Mo., ALR
LAPSLEY, ROBERT M.(1902) Masonic Temple, Keokuk, Ia., OALR
*LARGE, S. H.(1904).....Hanna Bldg., Cleveland, O., ALR
*LARKIN, BERNARD J.(1916)
Hume-Mansur Bldg., Indianapolis, Ind., Op.
LARSEN, CARL L.(1916)
1027 Lowry Bldg., St. Paul, Minn., OALR
LARUE, C. L.(1922).....Highland Clinic, Shreveport, La., OALR
LASALLE, ALBERT(1924)
3439 St. Denis St., Montreal, Can., OALR
LASSEN, FRITZ(1915).....Pueblo Clinic, Pueblo, Colo., ALR
LA VIGNE, A. A....471 Park Ave., New York City, N. Y., OALR
LAWWILL, STEWART(1928)
801 Chat. Bk. Bldg., Chattanooga, Tenn., OALR
LAWRENCE, GRANVILLE A.,
Medical Arts Bldg., Philadelphia, Pa., Op.

LEA, JOS. A.....1125 Rialto Bldg., Kansas City, Mo., ALR
*LEAVY, C. A.....Metropolitan Bldg., St. Louis, Mo., ALR
LEBENSOHN, JAMES E.(1927)
25 E. Washington, Chicago, Ill., OALR
LE COMPTE, E. D.(1927)
627 Boston Bldg., Salt Lake City, Utah., OALR
LEDERER, FRANCIS L.(1925)
30 N. Michigan Blvd., Chicago, Ill., ALR
LEECH, J. W.....369 Broad St., Providence, R. I., OALR
LEFLER, ANNA B.(1918)
605 S. Broadway, Los Angeles, Cal., OALR
LEHAN, JAMES W.....Park Place-8th St., Greeley, Colo., OALR
LEJEUNE, F. E.(1927)
632 Maison Blanche Bldg., New Orleans, La., ALR
LEMERE, H. B.(1912)...Medical Arts Bldg., Omaha, Neb., OALR
LEMOINE, A. N.(1920) 1118 Rialto Bldg., Kansas City, Mo., Op.
LENKER, JOHN N.(1908)..1020 Huron Rd., Cleveland, O., ALR
LENT, E. J.....122 N. Lafayette Blvd., South Bend, Ind., OALR
*LERNER, MACY LEVI(1923)
332 Park Ave., Rochester, N. Y., Op.
*LESTER, HARRY S.(1909)..420 Murray Bldg., Streator, Ill., OALR
*LEVENGOOD, H. W.(1927)
327 Wilshire Blvd., Santa Monica, Cal., OALR
*LEVY, LOUIS(1916)..1052 Madison Ave., Memphis, Tenn., ALR
*LEVY, ROBERT(1896)...Metropolitan Bldg., Denver, Colo., ALR
*LEWIS, A. C.(1916)....Exchange Bldg., Memphis, Tenn., OALR
LEWIS, EUGENE R.(1926)
Roosevelt Bldg., Los Angeles, Cal., ALR
LEWIS, PHILIP MERIWHETHER(1928)
Exchange Bldg., Memphis, Tenn., OALR
LEWIS, FIELDING O.....259 S. 17th St., Philadelphia, Pa., ALR
*LEWIS, F. PARK.....454 Franklin St., Buffalo, N. Y., Op.
*LEWIS, GEORGE A.(1920).....Roundup, Mont., OALR
LEWIS, J. D.(1921)..1534 Estado St., Santa Barbara, Cal., OALR
LEWIS, J. P.(1915).....543 Boylston St., Boston, Mass., ALR
LEWIS, WILLIAM W.(1921)
836 Lowry Bldg., St. Paul, Minn., OALR
LEYDA, JAMES H.(1927)
406 Metropolitan Bldg., Denver, Colo., ALR
LICHTENBERG, J. S.(1906) Rialto Bldg., Kansas City, Mo., Op.
LIDIKAY, C. J.(1920)..903 N. 7th St., Kansas City, Kans., OALR
*LIERLE, DEAN MCALLISTER(1928)
University Hosp., Iowa City, Ia., ALR
LIFSCHUTZ, JACOB(1925)
310 S. Michigan Ave., Chicago, Ill., OALR
*LILLIE, HAROLD I.....Mayo Clinic, Rochester, Minn., ALR
LINN, E. G.(1913)...714 Equitable Bldg., Des Moines, Ia., OALR
*LINN, J. G.(1919)
7026 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*LISCHKOFF, M. A.(1913)....Blount Bldg., Pensacola, Fla., OALR

LITCHFIELD, J. T.(1921)
609 Yeates Bldg., Minneapolis, Minn., OALR
LLOYD, A. W.(1915)...804 Lloyd Bldg., Hammond, Ind., OALR
LLOYD, RALPH I.(1925)..14 Eighth Ave., Brooklyn, N. Y., Op.
LOEB, CLARENCE(1907)
1054 Tower Rd., Hubbard Woods, Ill., Op.
LOFTUS, JOHN EDWARD(1921)
605 Medical Arts Bldg., Philadelphia, Pa., ALR
LOKEY, H. M.....Medical Arts Bldg., Atlanta, Ga., OALR
LOMBARDO, MELCHIORE(1923)
Medical Arts Bldg., Brooklyn, N. Y., Op.
*LOOMIS, E. A.(1916) Donaldson Bldg., Minneapolis, Minn., OALR
LOOPER, EDWARD ANDERSON(1924)
605 Park Ave., Baltimore, Md., OALR
LORIE, ALVIN(1920)..Commerce Bldg., Kansas City, Mo., ALR
LORING, J. B.....25 E. Washington St., Chicago, Ill., OALR
LOVE, JOHN G.(1920).....Ilgenfritz Bldg., Sedalia, Mo., OALR
LOWRY, B. W.(1925) 408 Citrus Exch. Bldg., Tampa, Fla., OALR
LUBMAN, MAX(1919)
17 E. 96th St., New York City, N. Y., ALR
LUCAS, HAROLD ROY(1928)
411 Selling Bldg., Portland, Ore., OALR
*LUEDDE, W. H.(1916) Metropolitan Bldg., St. Louis, Mo., Op.
LUKENS, CHARLES(1906)....316 Michigan St., Toledo, O., OALR
LUKENS, ROBT. McD...1923 Spruce St., Philadelphia, Pa., ALR
LUNDON, A. E.(1927)
1501 Crescent St., Montreal, Canada, ALR
LUPTON, IRVING MARTIN(1928)
Med. Arts Bldg., Portland, Ore., OALR
*LUX, PAUL(1925) 409 Commerce Bldg., Kansas City, Mo., ALR
LYLE, DONALD J.(1927).....19 W. 7th St., Cincinnati, O., OALR
*LYMAN, HARRY W.(1923)
700 Carleton Bldg., St. Louis, Mo., ALR
*LYNCH, R. C.(1913)
Maison Blanche Bldg., New Orleans, La., ALR
LYONS, H. R.....30 N. Michigan Blvd., Chicago, Ill., ALR

M

MCALESTER, ANDREW W., JR.,
625 Bryant Bldg., Kansas City, Mo., Op.
*MCALLISTER, J. C.(1910)...10 S. Mill St., Ridgeway, Pa., OALR
MCBEAN, GEO. M.....58 E. Washington St., Chicago, Ill., AL
MCBRIDE, W. O.(1917)
2906 S. Fairfield Ave., Ft. Wayne, Ind., OALR
*MCCANNELL, A. D.....20 Central Ave., Minot, N. D., OALR
MCCARTNEY, OLIVER P.(1920)
707 Huron Bldg., Kansas City, Kan., OALR
MCCARTY, VIRGIL W..800 Rialto Bldg., Kansas City., Mo.

McCaskey, C. H.,
Continental Bank Bldg., Indianapolis, Ind., ALR
McCaw, John A.....418 Majestic Bldg., Denver, Colo., Op.
McClellan, W. E.(1927)
101 Haywood St., Asheville, N. C., OALR
McClelland, Carl C.(1923)
1857 David Whitney Bldg., Detroit, Mich., OALR
McConnell, T. E.
Logan Trust Bldg., New Kensington, Pa., OALR
McCool, J. L.(1911)...909 Stevens Bldg., Portland, Ore., Op.
McCoy, J.....730 Fifth Ave., New York City, N. Y., ALR
McCready, J. H.....121 University Pl., Pittsburgh, Pa., ALR
McCulloch, R. J.(1920)..102 College St., Toronto, Can., OALR
*McDannald, C. E.(1927)
100 W. 59th St., New York City, N. Y., Op.
*McDermott, John J.(1924)
1871 Niles Ave., St. Joseph, Mich., OALR
McDiarmid, H. Oliver(1918)
35 Clement Block, Brandon, Manitoba, Can., OALR
McDonald, C. L.....964 Rose Bldg., Cleveland, O., OALR
McDonald, Dennis J.(1926)
17 E. 38th St., New York City, N. Y., ALR
McDowell, N. D.(1914)
275 Alexander St., Rochester, N. Y., OALR
McFarlane, Murray....190 Bloor St., E., Toronto, Can., OALR
McGinnis, Edwin...104 S. Michigan Blvd., Chicago, Ill., ALR
McGivern, C. S.(1922)
101 S. Indiana Ave., Atlantic City, N. J., ALR
McGuire, H. H....105 N. Braddock St., Winchester, Va., OALR
*McHenry, D. D.(1912)
604 Medical Arts Bldg., Oklahoma City, Okla., OALR
McKee, Claud W.(1917).Coulter Bldg., Greensburg, Pa., OALR
*McKee, Geo. J.(1922)
Westinghouse Bldg., Pittsburgh, Pa., ALR
*McKee, Joseph Wallace(1928)
1222 Rialto Bldg., Kansas City, Mo., Op.
McKee, Otto S.....131 Linwood Ave., Buffalo, N. Y., ALR
McKee, S. Hanford(1910)
1528 Crescent St., Montreal, Can., Op.
McKelvey, A. D.....160 Bloor St., W., Toronto, Can., ALR
McKennan, Jas. W.
514 Washington Trust Bldg., Washington, Pa., Op.
*McKeown, Elmer E...Metropolitan Bldg., Denver, Colo., OALR
*McKinney, A. R.(1922)
704 Second Natl. Bk. Bldg., Saginaw, Mich., OALR
McKinney, R.....1052 Madison Ave., Memphis, Tenn., ALR
McLaughlin, P.A.,
71 S. Washington St., Wilkes-Barre, Pa., OALR
McLaurin, John G.(1926)
1302 Medical Arts Bldg., Dallas, Texas, OALR

McLOONE, JOHN J.(1926) 611 Heard Bldg., Phoenix, Ariz., OALR
*McMAHON, BERNARD JOHN(1926)
Missouri Theatre Bldg., St. Louis, Mo., ALR
McMURRAY, J. B.,
Washington Trust Bldg., Washington, Pa., OALR
*McNALLY, WILLIAM JAMES
746 Sherbrooke St., W., Montreal, Canada, ALR
*McNICHOLS, W. A.(1925)
Dixon Nat'l. Bk. Bldg., Dixon, Ill., OALR
*McREYNOLDS, G. S.....State Bank Bldg., Temple, Tex., ALR
*McREYNOLDS, JOHN O.,
907 Mercantile Bk. Bldg., Dallas, Tex., OALR
McWILLIAMS, CHAS. A.(1927).....Gulfport, Miss., ALR
MACFARLANE, P. B.
152 James St., S., Hamilton, Ont., Can., OALR
MACKENZIE, ALICE V.(1919)
1724 Spruce St., Philadelphia, Pa., OALR
MACKENZIE, GEO. W.(1912)
1724 Spruce St., Philadelphia, Pa., OALR
MacLACHLAN, A. A.....May Bldg., Pittsburgh, Pa., ALR
MacMILLAN, J. A.(1925)
1429 Stanley St., Montreal, Canada, Op.
MACNIE, J. S.....74 S. 11th St., Minneapolis, Minn., OALR
MADDEN, REED.....Allen Bldg., Xenia, O., OALR
*MAGGARD, D. I.(1920).715 Orpheum Bldg., Wichita, Kan., OALR
MAGHY, CHAS. A.(1926)
1136 W. 6th St., Los Angeles, Cal., Op.
MAHONEY, PAUL LEO(1928)
Donaghey Bldg., Little Rock, Ark., ALR
MAIDEN, S. D.(1918)
403 Bennett Bldg., Council Bluffs, Ia., OALR
MAIRE, LEWIS E.(1906).710 Kresge Bldg., Detroit, Mich., OALR
MALONEY, THOS. J....1032 Lowry Bldg., St. Paul, Minn., OALR
*MANN, R. H. T.(1913)
State Natl. Bk. Bldg., Texarkana, Tex., OALR
*MARCH, CLARA A.(1922)
465 Ashland Ave., Buffalo, N. Y., Op.
MARKEL, J. CLYDE...Westinghouse Bldg., Pittsburgh, Pa., Op.
*MARKS, SAMUEL BLACKBURN(1928)
Sec. Trust Bldg., Lexington, Ky., ALR
*MARLOW, SEARLE B.(1924)
1004 State Tower Bldg., Syracuse, N. Y., Op.
MARQUIS, GEO. P.....30 N. Michigan Blvd., Chicago, Ill., ALR
MARSHALL, GEORGE GUERIN..Gryphon Bldg., Rutland, Vt., OALR
MARSHALL, WATSON..121 University Pl., Pittsburgh, Pa., ALR
*MASON, ROY EARL(1928)
422 Frisco Bldg., St. Louis, Mo., Op.
*MASTERS, ROBERT JOHN(1928)
601 Hume-Mansur Bldg., Indianapolis, Ind., Op.

MATHENY, R. C.
405 Bk. of Galesburg Bldg., Galesburg, Ill., OALR
MATHESON, J. P. (1915)..6 West 7th St, Charlotte, N. C., ALR
MATTHEWS, JUSTUS
1021 Metropolitan Bk. Bldg., Minneapolis, Minn., LR
MAUDLIN, L. O.
Chamber of Commerce Bldg., Greenville, S. C., OALR
MAUMENEE, A. E....711 Empire Bldg., Birmingham, Ala., OALR
MAXEY, E. E. (1911)..609 Becker Bldg., Aberdeen, Wash., OALR
MAY, CHAS. H. (1904)
698 Madison Ave., New York City, N. Y., Op.
MAY, J. V. (1915).....1703 Main St., Marinette, Wis., OALR
*MAY, JAMES W.....Huron Bldg., Kansas City, Kan., Op.
MAYBAUM, J. L. (1916)
1079 Park Ave., New York City, N. Y., ALR
*MAYER, LEO LOEB, (1928)
900 Carleton Bldg., St. Louis, Mo., Op.
MEANOR, WM. C., 619 Westinghouse Bldg., Pittsburgh, Pa., Op.
MEIERHOF, E. L. (1912)
12 E. 86th St., New York City, N. Y., OALR
MELLINGER, WM. J. (1927)
120 E. Sola St., Santa Barbara, Cal., ALR
MERCER, RAY (1920).....W. C. U. Bldg., Quincy, Ill., OALR
MERCER, R. E.....David Whitney Bldg., Detroit, Mich., LR
MERKEL, CHARLES CARSON (1928)
1409 David Whitney Bldg., Detroit, Mich., ALR
MERRILL, H. G. (1917)
33 East 2nd St., South, Provo, Utah, OALR
MERRILL, W. HOWE (1906)
301 Essex St., Lawrence, Mass., OALR
*MERTINS, PAUL STEARNS (1928)
1115 Bell Bldg., Montgomery, Ala., OALR
*MESSIER, A. E. (1925)....390 Main St., Worcester, Mass., OALR
MESSINGER, H. C.....210 Angell St., Providence, R. I., Op.
METZ, ROY B.....1214 Guardian Bldg., Cleveland, O., Op.
METZENBAUM, MYRON (1912)...Rose Bldg., Cleveland, O., ALR
*MEYER, SAMUEL JAMES (1928)
58 E. Washington St., Chicago, Ill., Op.
*MIDDLETON, A. B.....Pontiac, Ill., OALR
*MIDDLETON, HARRY E. (1920).....Times Bldg., Alton, Ill., OALR
MILLER, ALICE R. (1919)
505 Guaranty Bldg., West Palm Beach, Fla., OALR
MILLER, C. E....305 Western Reserve Bldg., Muncie, Ind., OALR
MILLER, CLIFTON M.
Stuart Circle Hospital, Richmond, Va., OALR
MILLER, E. B.....2008 Walnut St., Philadelphia, Pa., Op.
MILLER, HUGH.....Chambers Bldg., Kansas City, Mo., OALR
*MILLER, VERNON B. (1928)
810 Metropolitan Bldg., St. Louis, Mo., ALR

*MILLETTE, J. W. . . . . Reibold Bldg., Dayton, O., OALR
MINER, S. G. (1896) . . . . 2641 E. Jefferson, Detroit, Mich., ALR
MINOR, C. L. . . . . First Natl. Bank Bldg., Springfield, O., OALR
MINOR, H. F. . . . . Randolph Bldg., Memphis, Tenn., OALR
MINSKY, HENRY (1926)
215 W. 88th St., New York City, N. Y., Op.
MINTON, WM. H. . First State Bank Bldg., St. Joseph, Mo., OALR
MITCHELL, E. K. . . 710 W. Lehigh Ave., Philadelphia, Pa., ALR
*MITHOEFER, W. . . . . 19 Garfield Pl., Cincinnati, O., ALR
MITTENDORF, A. D. . 399 Park Ave., New York City, N. Y., Op.
MITTENDORF, W. KEITH,
115 E. 53rd St., New York City, N. Y., Op.
*MOISE, ALLYN B. (1918)
Medical Arts Bldg., Shreveport, La., ALR
*MOLT, WM. F. (1911)
529 Bankers Trust Bldg., Indianapolis, Ind., ALR
MONAGHAN, D. G. . . . . 535 Majestic Bldg., Denver, Colo., Op.
MONAHAN, J. A. (1913) . . 181 Chestnut St., Clinton, Mass., OALR
MONOSMITH, O. B. (1912) . . . . . O. P. S. Bldg., Lorain, O., OALR
MONSON, S. H. . . . . 1322 B. F. Keith Bldg., Cleveland, O., OALR
*MONTGOMERY, DANIEL CAMERON (1928) . . Greenville, Miss., OALR
MOORE, G. A. (1915) . . . . . Main St., Palmer, Mass., OALR
MOORE, GEO. H. (1919) . . . . Schuylkill Bldg., Pottsville, Pa., OALR
MOORE, JOHN D. (1925)
6 Washington St., Bloomfield, N. J., OALR
MOORE, ROY SEELEY (1928)
405 E. Fayette St., Syracuse, N. Y., ALR
*MOORE, T. K. . . 416 Central Sav. & Trust Bldg., Akron, O., OALR
*MOORE, T. W. (1901) 1050 Fifth Ave., Huntington, W. Va., OALR
MOORE, WILLIAM FREDERICK
255 S. 16th St., Philadelphia, Pa., ALR
MORGAN, JAMES A. (1916)
48 Young Bldg., Honolulu, T. Hawaii, OALR
MORIARTY, RICHARD W. (1928)
38 S. Broadway, White Plains, N. Y., OALR
MORRIS, A. G. (1918) . 303 Alexander St., Rochester, N. Y., ALR
MORRISON, WM. WALLACE (1926)
39 E. 50th St., New York City, N. Y., ALR
MORSE, A. W. . . . . 507 Phoenix Bldg., Butte, Mont., OALR
*MORSMAN, L. W. (1920) . 205 Power Bldg., Hibbing, Minn., Op.
MORTON, HOWARD MCI.
418 Insurance Exchange Bldg., Minneapolis, Minn., Op.
MORTON, J. B. (1924) . . 90 E. Warren Ave., Detroit, Mich., OALR
*MOSHER, H. P. . . . . 828 Beacon St., Boston, Mass., ALR
*MOSS, HARVEY E. . . . . 1321 Rialto Bldg., Kansas City, Mo., OALR
MOTT, JOHN S. . . . . 214 Argyle Bldg., Kansas City, Mo., OALR
MOULTON, EVERETT C
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
*MOULTON, H. (1900)
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR

*MULLIN, W. V. (1913)
Cleveland Clinic, Euclid Ave. & 93rd, Cleveland, O., ALR
MULSOW, J. E. (1921)
230 Wisconsin Ave., W., Milwaukee, Wis., OALR
*MUNCASTER, S. B. 1026 16th St., N. W., Washington, D. C., Op.
MUNCY, WM. M. (1914) . . 162 Angell St., Providence, R. I., OALR
MUNDT, G. H. (1912) . . 25 E. Washington St., Chicago, Ill., OALR
MURDOCK, J. F. . . . . Bessemer Bldg., Pittsburgh, Pa., Op.
MURPHY, J. G. . . . . Murchison Bldg., Wilmington, N. C., OALR
MURPHY, W. E. . . . . 7th and Race Sts., Cincinnati, O., OALR
MURRAY, A. N. . . . . 4753 Broadway, Chicago, Ill., OALR
MUSSUN, W. G. (1924) . . . . 610 Hanna Bldg., Cleveland, O., ALR
MYERS, ELMER A.,
407 Board of Trade Bldg., Superior, Wis., OALR
*MYERS, EUGENE LEE (1926)
207 Wall Bldg., St. Louis, Mo., ALR
*MYERS, JOHN L. (1920)
815 Shukert Bldg., Kansas City, Mo., OALR
MYERSON, M. C. (1923) . . . . 30 E. 4th St., Brooklyn, N. Y., ALR

N

NADEAU, E. G. . . . Bellin-Buchanan Bldg., Green Bay, Wis., OALR
*NAFTZGER, J. B. . . . . Davidson Bldg., Sioux City, Ia., OALR
NANCE, WILLIS O. . . . . 30 N. Michigan Blvd., Chicago, Ill., OALR
NEEPER, EDW. R.,
EX. Nat'l. Bank Bldg., Colorado Springs, Colo., Op.
NEHER, EDWIN MANSON (1926)
Boston Bldg., Salt Lake City, Utah, Op.
NEIGHBORS, CLARENCE A.,
R. F. D. No. 2, Box 87, Anaheim, Cal., OALR
NELSON, CHAS. F. . . . . 606 Schofield Bldg., Cleveland, O., OALR
NELSON, LOUIS A. . . . . 830 Lowry Bldg., St. Paul, Minn., Op.
NELSON, LLOYD L. (1923)
Ommerman Bldg., Norfolk, Neb., OALR
NEUMANN, WILLIAM HENRY (1928)
1005 N. 8th St., Sheboygan, Wis., OALR
*NEW, GORDON B. . . . . Mayo Clinic, Rochester, Minn., L
NEWCOMB, JOHN R.,
410 Hume-Mansur Bldg., Indianapolis, Ind., Op.
NEWELL, WM. S. . 1029 Vermont Ave., Washington, D. C., Op.
*NEWHART, HORACE (1905)
Donaldson Bldg., Minneapolis, Minn., ALR
NEWTON, F. H. (1925) . . 907 Mercantile Bldg., Dallas, Tex., OALR
NEWTON, L. A. (1919)
609 Medical Arts Bldg., Oklahoma City, Okla., OALR
NISSELSON, MAX. . . . . 754 Beck St., New York City, N. Y., ALR
NOONAN, W. J. (1905) . . . . . 4753 Broadway, Chicago, Ill., OALR

NORCROSS, E. R. (1924) . 30 N. Michigan Ave., Chicago, Ill., ALR
NORTHCOTT, THOMAS A. (1926)
17 E. 38th St., New York City, N. Y., Op.
NOVAK, FRANK J., JR. . 30 N. Michigan Blvd., Chicago, Ill., ALR
NUTTER, C. F. . . . . 16 Amherst St., Nashua, N. H., Op.

O

OBERG, C. M. . . . . Syndicate Bldg., Minneapolis, Minn., OALR
O'BRIEN, C. S. (1924) . . University Hospital, Iowa City, Ia., Op.
*O'BRIEN, STEVE A. (1916)
1st Nat'l. Bank Bldg., Mason City, Ia., OALR
O'CONNOR, D. F. (1918) . . . . . 671 Broad St., Newark, N. J., OALR
O'CONNOR, RODERIC P. . . 1904 Franklin St., Oakland, Cal., Op.
OERTEL, T. E. . . . . 638 Green St., Augusta, Ga., OALR
OESTERLING, H. E. (1922) . . . . . Box 454, Winter Park, Fla., OALR
OFFUTT, W. N. (1905)
406 Security Trust Bldg., Lexington, Ky., OALR
*OLSHO, S. L. (1912) . . . . . 235 S. 15th St., Philadelphia, Pa., OALR
ORCUTT, D. C. (1912) . . 30 N. Michigan Ave., Chicago, Ill., Op.
O'REILLY, CHARLES A. (1911)
521 Med. Arts Bldg., Philadelphia, Pa., ALR
*O'REILLY, W. F. (1913) . . 44 S. Common St., Lynn, Mass., OALR
*O'ROURKE, D. H. (1927) . 217 Imperial Bldg., Denver, Colo., Op.
ORTON, HENRY B. . . . . 24 Commerce St., Newark, N. J., ALR
O'SULLIVAN, T. J. (1927) . . . . . 10 Pine St., Portland, Me., ALR
OVERMAN, F. V. . . Hume-Mansur Bldg., Indianapolis, Ind., ALR
OWEN, F. S. . . . . Medical Arts Bldg., Omaha, Neb., OALR

P

PACKARD, LOUIS (1926) . . . . . Denver, Colo., OALR
PAGE, LAFAYETTE. Hume-Mansur Bldg., Indianapolis, Ind., ALR
*PAINTER, A. M. (1918)
418 Bryant Bldg., Kansas City, Mo., ALR
PALMER, ARTHUR (1926)
667 Madison Ave., New York City, N. Y., ALR
PALMER, BASCOM HEADEN (1928)
502 Huntington Bldg., Miami, Fla., OALR
PALMER, EDW. M. (1919) 629 Beacon Bldg., Wichita, Kan., OALR
*PALMER, FRANK ERNEST (1928) . . . . . Sterling, Colo., OALR
PARK, J. W. (1910) . . . . . 32 N. Second St., Harrisburg, Pa., OALR
*PARKER, WALTER R. . David Whitney Bldg., Detroit, Mich., Op.
PARRISH, MAJ. ROBERT E., M. C., U. S. A.,
Wm. Beaumont Gen. Hospital, El Paso, Tex., ALR
PARSONS, J. G. . 313 Bank Electric Bldg., Lewistown, Mont., OALR
PATTERSON, ELLEN J. (1917)
121 University Pl., Pittsburgh, Pa., ALR
PATTERSON, JAS. A. (1904)
Burns Bldg., Colorado Springs, Colo., OALR

*PATTERSON, W. E.....74 S. 11th St., Minneapolis, Minn., OALR
*PATTON, J. M.(1910)...Medical Arts Bldg., Omaha, Neb., Op.
PAYNE, A. L.(1908).Opera House Bldg., Eau Claire, Wis., OALR
*PAYNE, R. J.(1925)
University Club Bldg., St. Louis, Mo., ALR
PEARLMAN, S. J.....180 N. Michigan Ave., Chicago, Ill., ALR
*PEARSON, WM. W.(1908)
417 Bankers Trust Bldg., Des Moines, Ia., OALR
PEELER, C. N.(1920).....W. 7th St., Charlotte, N. C., OALR
PEIRCE, HOWARD W.(1923)
704 Med. Arts Bldg., Detroit, Mich., ALR
*PEMBER, AUBREY H.(1926)
500 W. Milwaukee St., Janesville, Wis., OALR
PENDEXTER, RALPH S.(1926)
1424 K St., N. W., Washington, D. C., Op.
PENTECOST, R. S.(1926)...184 Bloor St., Toronto, Canada, OALR
*PETER, L. C.(1914)
N. E. cor. Chestnut & 20th Sts., Philadelphia, Pa., Op.
PETERMAN, H. E.(1912)
512 Cathedral St., Baltimore, Md., OALR
PETERSON, R. A.(1920)
150 Fifth Ave., New York City., N. Y., OALR
PFAFFLIN, C. A.
445 Bankers Trust Bldg., Indianapolis, Ind., OALR
PFINGST, A. O.....712 Brown Bldg., Louisville, Ky., Op.
*PFINGSTEN, C. F.(1916)
820 Metropolitan Bldg., St. Louis, Mo., ALR
*PHELPS, KENNETH A.(1920)
730 La Salle Bldg., Minneapolis, Minn., OALR
PHILLIPS, W. C.....30 E. 58th St., New York City, ALR
PHILLIP, WM. LINTON..131 Linwood Ave., Buffalo, N. Y., Op.
PHINNEY, F. D.....22 W. 7th St., Cincinnati, O., OALR
PIPER, ARTHUR SYLVESTER(1928)
Enid Clinic, Enid, Okla., OALR
*PITKIN, C. E.....Osborn Bldg., Cleveland, O., ALR
PLUMER, JOHN SHAFFER(1928)
121 University Pl., Pittsburgh, Pa., Op.
*POLLOCK, H. L.(1911)....2551 N. Clark St., Chicago, Ill., ALR
PONTIUS, P. J.....1831 Chestnut St., Philadelphia, Pa., Op.
POOS, E. E.(1927)..13300 Woodward Ave., Detroit, Mich., OALR
*POOS, GROVER HEWITT(1928)
301 Univ. Club Bldg., St. Louis, Mo., OALR
PORTER, E. H.(1912).....85 Madison St., Tiffin, O., OALR
*PORTER, JESSE B.(1920).....415 S. 2nd St., Elkhart, Ind., OALR
PORTER, L. B.(1913)....199 Thayer St., Providence, R. I., OALR
*POST, LAWRENCE T.(1920)
520 Metropolitan Bldg., St. Louis, Mo., Op.
*POST, M. HAYWARD(1918)
520 Metropolitan Bldg., St. Louis, Mo., Op.

POST, WINFRED B. (1921)
First National Bank Bldg., Carthage, Mo., OALR
*POTTER, GEO. B. (1921)..Medical Arts Bldg., Omaha, Neb., OALR
POTTER, L. F.....68 Walnut St., New Bedford, Mass., OALR
POTTER, PETER (1918)...125 W. Granite St., Butte, Mont., OALR
*POTTER, W. W. (1927)....Medical Bldg., Knoxville, Tenn., OALR
POTTS, J. B.....Medical Arts Bldg., Omaha, Neb., ALR
*POWELL LYLE STEPHENSON (1928)
813 Mass. St., Lawrence, Kan., OALR
*POWER, EVERETT (1916)
Central Nat'l. Bank Bldg., Carthage, Mo., OALR
PRANGEN, AVERY DE H. (1919)
Mayo Clinic, Rochester, Minn., Op.
*PRATT, FRED J. (1912)
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
PRATT, JOHN A. (1910)
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
PRENDERGAST, D. A.....850 Hanna Bldg., Cleveland, O., OALR
PRICE, NORMAN W. (1908)
445 3rd St., Niagara Falls, N. Y., OALR
PRINCE, ALEXANDER L. (1928)
36 Pearl St., Hartford, Conn., Op.
*PROETZ, A. W. (1923)....Beaumont Bldg., St. Louis, Mo., ALR
PROUD, WILLARD C. (1920)
Phys. & Surg. Bldg., St. Joseph, Mo., OALR
PROUT, A. W. (1920).....370 E. Town St., Columbus, O., OALR
PUTNAM, F. I.....Smithson Bldg., Ellensburg, Wash.
PYLE, WALLACE (1915).15 Exchange Pl., Jersey City, N. J., OALR

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*QUALLS, HARLEY WINFIELD (1928)
1501 Exchange Bldg., Memphis, Tenn., OALR
QUINBY, WM. O'G. (1919)...14 James St., Newark, N. J., OALR
QUINCY, J. E. (1927)
270 Commonwealth Ave., Boston, Mass., ALR
QUITTNER, S. S.....3912 Prospect Ave., Cleveland, O., OALR

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RADCLIFFE, MCCLUNEY,
1906 Chestnut St., Philadelphia, Pa., Op.
*RALLS, C. T. (1916)..1st Nat'l. Bank Bldg., Winfield, Kas., OALR
RALSTON, WALLACE (1918)
1300 Walker Ave., Houston, Tex., Op.
RAMSEY, G. S. (1927)
1496 Mountain St., Montreal, Canada, Op.
*RANDALL, HIRAM (1928)..81 Main St., Binghamton, N. Y., ALR
*RAVDIN, B. D....Citizens Natl. Bk. Bldg., Evansville, Ind., OALR
RAVDIN, M.....Citizens Natl. Bk. Bldg., Evansville, Ind., OALR

RAY, VICTOR (1901) .....19 W. 7th St., Cincinnati, O., Op.
REAVES, CHAS. R.,
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
REAVES, ROBERT GRISHAM (1928) .....Knoxville, Tenn., OALR
*REAVES, W. P. (1913)
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
REBUCK, C. S. .....412 N. Third St., Harrisburg, Pa., OALR
*RECTOR, A. E. (1915)
112 W. College Ave., Appleton, Wis., OALR
REEDER, J. E. (1916) ..224 Davidson Bldg., Sioux City, Ia., OALR
REESE, ALGERNON B. (1928)
50 W. 52nd St., New York City, N. Y., Op
REESE, MONROE D. (1926) ...123 N. 8th St., Lebanon, Pa., OALR
REESE, WARREN S. (1921) 230 S. 21st St., Philadelphia, Pa., Op.
*REGER, H. S. (1908) .....15 E. Fourth, Jamestown, N. Y., OALR
REICHE, O. C. (1916) ....Markle Bk. Bldg., Hazelton, Pa., OALR
*REIM, HUGO. .....417 Metropolitan Bldg., St. Louis, Mo., Op.
REINEKE, GEO. F. (1926) .....New Ulm, Minn., OALR
RENAUD, G. L. ....510 Joseph Campan Ave., Detroit, Mich., OALR
RENNER, W. S. (1900) ..341 Linwood Ave., Buffalo, N. Y., ALR
REPASS, R. E. (1920)
Lincoln Rd. and Washington Ave., Miami Beach., Fla. ALR
REULING, F. H. (1924) .....Black Bldg., Waterloo, Ia., OALR
*REYNOLDS, H. G. (1909)
City Natl. Bk. Bldg., Paducah, Ky., OALR
*REYNOLDS, J. S. (1918) .La Salle Bldg., Minneapolis, Minn., OALR
*RHODE, SOLON LAFAYETTE (1928)
238 N. 6th St., Reading, Pa., OALR
RICE, CLARENCE PRENTICE. .....Wahpeton, N. D., OALR
RICE, G. B. (1916) 270 Commonwealth Ave., Boston, Mass., ALR
RICE, JOHN E. (1924) .....390 Main St., Worcester, Mass., OALR
RICE, R. H. (1915) .....331 Grove St., Milwaukee, Wis., OALR
RICHARDS, L. G. (1924)
270 Commonwealth Ave., Boston, Mass., ALR
RICHARDSON, CHAS. W.,
1337 Connecticut Ave., Washington, D. C., ALR
*RICHARDSON, S. A. (1925)
111 W. Adams St., Jacksonville, Fla., Op.
RICHIE, L. T. (1916) ...McCormick Bldg., Trinidad, Colo., OALR
RIDEOUT, W. J. .....701 State Bk. Bldg., Freeport, Ill., OALR
*RIDPATH, ROBT. F.,
N. E. cor. 18th & Chestnut Sts., Philadelphia, Pa., RL
RIEGER, W. H. (1924) .....Hanna Bldg., Cleveland, O., ALR
RINDLAUB, ELIZABETH P. .....620 Front St., Fargo, N. D., OALR
RINDLAUB, JOHN H. .....620 Front St., Fargo, N. D., OALR
RINDLAUB, MARTIN P. .....620 Front St., Fargo, N. D., OALR
RINGLE, CHAS. A. (1906) .....Box 429, Greeley, Colo., ALR
RINGUEBERG, E. N. S. (1905)
13 Main St., Lockport, N. Y., Op.
RISDON, E. F. (1924) ....30 Avenue Rd., Toronto, Canada, ALR

*Risser, Edward D. (1928)
310 Choate Bldg., Winona, Minn., OALR
*Roberts, S. E. (1916)
1810 Federal Reserve Bank Bldg., Kansas City, Mo., ALR
Roberts, Wm. H. . . . . 65 N. Madison Ave., Pasadena, Cal., Op.
*Robertson, Edw. N. (1918)
132½ W. 6th St., Concordia, Kan., OALR
Robertson, F. D. . . . . Ashton Bldg., Grand Rapids, Mich., OALR
Robinson, H. T. (1916) . . Medical Bldg., Cumberland, Md., OALR
*Robinson, R. E. (1920)
205½ E. Bremer Ave., Waverly, Ia., OALR
Rogers, A. S. . . . . 105 S. Jefferson Ave., Saginaw, Mich., OALR
Rogers, Robert Merriam (1928)
680 St. Johns Pl., Brooklyn, N. Y., Op.
Rogers, J. T. . . . . Med. Arts Bldg., Montreal, Can., ALR
Roller, Louis A. (1920)
446 Paris Ave., S. E., Grand Rapids, Mich., Retired
Rommel, John C. . . . . 3701 N. Broad St., Philadelphia, Pa., Op.
*Rooker, A. M. (1920)
225 Gluck Bldg., Niagara Falls, N. Y., ALR
Roost, F. H. (1914) . . . . . Sioux City, Ia., OALR
Rosenbaum, J.
1396 St. Catherine's St., W., Montreal, Can., Op.
Roth, Albert W. (1920)
Medical Arts Bldg., Tulsa, Okla., OALR
Rothschild, Harold J. . . 239 Lowry Bldg., St. Paul, Minn., OALR
Rott, O. M. (1915) . . . . . Paulsen Bldg., Spokane, Wash., OALR
Row, Geo. S. (1911)
906 Hume-Mansur Bldg., Indianapolis, Ind., Op.
Rowland, John F. (1911)
Thompson Bldg., Hot Springs, Ark., OALR
Rowland, W. D. (1923) . 220 Clarendon St., Boston, Mass., Op.
*Rubendall, C. (1915) . . Medical Arts Bldg., Omaha, Neb., OALR
*Ruby, F. M. . . . . Hibbing, Minn., OALR
*Rucker, E. W. Jr. (1916)
926 Woodward Bldg., Birmingham, Ala., ALR
Rush, C. C. . . . 907 U. S. Natl. Bk. Bldg., Johnstown, Pa., OALR
Russell, E. L. (1920)
700 Argyle Bldg., Kansas City, Mo., OALR
Russell, J. A. M. (1922) . . . . . 813 Sassafras St., Erie, Pa., ALR
*Russell, R. E. (1924) 601 Pioneer Bk. Bldg., Waterloo, Ia., OALR
Rust, E. G. . . . . Hanna Bldg., Cleveland, O., Op.A
*Rutherford, C. W. (1925)
University Hospital, Dept. Ophth., Iowa City, Iowa, Op.
*Ryan, L. M. (1927) . . . . . Wall Bldg., St. Louis; Mo., ALR
Rychener, Ralph Orlando (1928)
1720 Exchange Bldg., Memphis, Tenn., Op.
Ryder, D. R. . . . . 151 Rock St., Fall River, Mass., ALR
*Ryerson, F. L. (1925) . . . . Fine Arts Bldg., Detroit, Mich., OALR

S

SACCO, ANTHONY (1920)
440 New York Ave., Union City, N. J., OALR
*SALINGER, SAMUEL (1920)
25 E. Washington St., Chicago, Ill., ALR
SALLANDER, F. W. (1919)
204 Commerce Bldg., Sioux City, Ia. OALR
SANDERSON, DAVID D. (1916) . . Funke Bldg., Lincoln, Neb., OALR
*SANDERSON, H. H. (1906)
David Whitney Bldg., Detroit, Mich., OALR
SANOR, DANIEL GALBREATH (1928)
206 E. State St., Columbus, O., ALR
SARTAIN, PAUL J. (1921)
2006 Walnut St., Philadelphia, Pa., Op.
SAUER, W. E. (1904) . . . . . Humboldt Bldg., St. Louis, Mo., ALR
SAUER, W. W. (1910) . . . . . Box 416, Marietta, O., OALR
SAVAGE, M. M (1927) . . . . 1720 Eutaw Pl., Baltimore, Md., OALR
*SAVERY, C. E. . . . . 126 N. Lafayette Blvd., South Bend, Ind., OALR
SCAL, JOSEPH COLEMAN (1926)
44 W. 74th St., New York City, N. Y., ALR
*SCALES, H. L. (1920)
Rorabaugh-Wiley Bldg., Hutchinson, Kas., OALR
SCALES, J. L. (1915)
Commercial Natl. Bk. Bldg., Shreveport, La., OALR
*SCALES, J. W. (1896) . . . . 204½ Main St., Pine Bluff, Ark., OALR
SCARLETT, HUNTER W. (1919)
230 S. 21st St., Philadelphia, Pa , Op.
*SCHALL, L. A. (1927)
520 Commonwealth Ave., Boston, Mass., Op
SCHARPPERT, N. L. (1916)
59 S. Washington St., Wilkes-Barre, Pa., OALR
*SCHENCK, C. P. (1920)
Medical Arts Bldg., Fort Worth, Tex., OALR
SCHILD, E. H. (1906) . . . . . Brant Bldg., Canton, O., Op.
SCHILLER, A. N. (1918)
249 West End Ave., New York City, N. Y., ALR
SCHLANSER, MAJOR A. E. (1921)
Walter Reed Gen. Hosp., Army Med. Center, Washington, D. C. . . . . OALR
SCHLEGEL, HERMAN T. (1916)
501 Third St., Wausau, Wis., OALR
SCHLINDWEIN, G. WM. . . . . 138 W. 9th St., Erie, Pa., OALR
SCHLIVEK, KAUFMAN (1925)
1016 Fifth Ave., New York City, N. Y., Op.
SCHNEIDEMAN, T. B. . 2045 Chestnut St., Philadelphia, Pa., Op.
*SCHNEIDER, ANTON S. (1928)
56 Court St., Plattsburg, N. Y., Op.A.
*SCHOLZ, R. P. . . . . Metropolitan Bldg., St. Louis, Mo., ALR
SCHRANT, J. H. (1916)
Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR

SCHULZE, E. C.(1926).1205 Med. Arts Bldg., Dallas, Tex., OALR
SCHURTER, M. A.(1924)
114 E. 7th St., Long Beach, Cal., OALR
SCHUSTER, FRANK P.(1927)
403 1st Natl. Bank Bldg., El Paso, Tex., OALR
SCHUSTER, S. A.(1925).1st Natl. Bk. Bldg., El Paso, Tex., ALR
SCHUTZ, W. H.(1921)....Bryant Bldg., Kansas City, Mo., OALR
SCHWARTZ, A. A.(1927)
203 W. 90th St., New York City, N. Y., ALR
*SCHWARTZ, FREDERICK O.(1919)
701 Metropolitan Bldg., St. Louis, Mo., Op.
*SCHWARTZ, WILLIAM A.(1928).....Phoenix, Ariz., OALR
SCHWEINITZ, GEO. E. DE(1905)
1705 Walnut St., Philadelphia, Pa., Op.
SCOTT, J. R.(1920).....Ottawa, Kan., OALR
SCOTT, MERLE E.(1926)..1st Natl. Bk. Bldg., Massillon, O., OALR
SCROGGS, J. J.(1921).....218 E. Third St., Beaver, Pa., Op.A
SEAMAN, GILBERT E....407 Wells Bldg., Milwaukee, Wis., OALR
*SEARCY, H. B.(1912).....2501 6th St., Tuscaloosa, Ala., OALR
*SEARS, W. H.(1910).....514 Penn St., Huntingdon, Pa., OALR
SEAY, D. E.....907 Mercantile Bk. Bldg., Dallas, Tex., OALR
SECOY, F. L.(1922)...301 Davidson Bldg., Sioux City, Ia., OALR
SEDGWICK, O. W.(1917).....Ionia, Mich., OALR
SEDWICK, W. A.....Republic Bldg., Denver, Colo., Op.
*SEEGMAN, SIMON(1918)...3401 5th Ave., Pittsburgh, Pa., ALR
SEELYE, W. K.(1920)..742 Stimson Bldg., Seattle, Wash., OALR
SELFRIDGE, G. L.....384 Post St., San Francisco, Cal., ALR
SELIGSTEIN, MILTON B.(1926)
801 Union and Planters Bk. Bldg., Memphis, Tenn., OALR
*SELLERS, L. M.(1920)....Medical Arts Bldg., Dallas, Tex., ALR
SENER, WALTER J.....Physicians Bldg., Philadelphia, Pa., Op.
SENSENEY, EUGENE TOWNER(1928)
316 Lister Bldg., St. Louis, Mo., ALR
SEWALL, E. C.(1910)
Stanford Univ. Hospital, San Francisco, Cal., ALR
*SEYDELL, E. M.(1916)
1030 First Natl. Bank Bldg., Wichita, Kas., ALR
*SHACKLETON, W. E.(1918)
2323 Prospect Ave., Cleveland, O., Op.
SHAFER, J. J.(1913).....Starks Bldg., Louisville, Ky., OALR
*SHAHAN, W. E....520 Metropolitan Bldg., St. Louis, Mo., Op.
SHANKLIN, E. M.....575 Hohman St., Hammond, Ind., OALR
SHANNON, C. E. G.....135 S. 17th St., Philadelphia, Pa., Op.
SHAPERO, I. M.(1919).289 Alexander St., Rochester, N.Y., OALR
*SHARP, BENJ. S.(1926)..341 Thayer St., Providence, R. I., OALR
SHARRETT, GEO. O.(1921)..119 Bedford, Cumberland, Md., OALR
SHASTID, T. H.(1913)
10th Ave. Med. Bldg., Duluth, Minn., Op.
SHAWHAN, GLENN E.(1913)Smallwood Bldg., Nampa,Ida., OALR
*SHEA, J. J.(1919)....1018 Madison Ave., Memphis, Tenn., ALR

*SHEAHAN, WM. L.(1926)
59 College St., New Haven, Conn., OALR
SHELLMAN, J. L.(1917)....Lowry Bldg., St. Paul, Minn., OALR
SHEMELEY, WM. G., JR., 1724 Spruce St., Philadelphia, Pa., OALR
SHERMAN, MAJ. A. ED., M.C., U. S. A.(1913)
Hdqts. Clover Field, Santa Monica, Cal., OALR
SHERMAN, ELBERT S.(1912).671 Broad St., Newark, N. J., Op.
*SHIELDS, J. M.(1915)264 Metropolitan Bldg., Denver,Colo., Op.
SHIELDS, LAWRENCE.....Allen Bldg., Xenia, O., ALR
SHIRAS, HOWARD H.(1920)
629 Guardian Bldg., Cleveland, O., Op.
*SHIREY, O. M.(1925).....Osborn Bldg., Cleveland, O., ALR
SHOEMAKER, FERDINAND, U. S. Vetersans' Hospital No. 67,
27th and Paseo, Kansas City, Mo., OALR
SHOEMAKER, S. A.....Bluffton, Ind., OALR
SHOEMAKER, W. A.(1896)
1006 Carleton Bldg., St. Louis, Mo., Op.
SHORT, Z. N.....Dugan-Stuart Bldg., Hot Springs, Ark., OALR
SHREVE, OWEN M.(1905).....162 W. 8th St., Erie, Pa., Op.
SHULTS, E. J. G.(1920).....Eldorado, Kansas, OALR
SHUMAN, G. H.(1917).....Park Bldg., Pittsburgh, Pa., Op.
SHUMATE, D. L.....500 Argyle Bldg., Kansas City, Mo., OALR
*SHURLY, BURT R.....62 Adams Ave., W., Detroit, Mich., ALR
SHUTE, D. K..1727 DeSales St., N.W., Washington, D. C., Op.
*SIEGEL, FRANCIS X.(1922)
904 Union Central Life Bldg., Cincinnati, O., OALR
SILBERNAGEL, C. E.(1918).283 E. State St., Columbus, O., OALR
SIMONDS, O. F.....Hanna Bldg., Cleveland, O., Op.
*SIMPSON, H. LEE(1921)
1409 David Whitney Bldg., Detroit, Mich., ALR
*SIMPSON, W. L.(1911)
Phys. & Surg. Bldg., Memphis, Tenn., ALR
SINAMARK, ANDREW(1928)
205 Power Bldg., Hibbing, Minn., OALR
SINGLETON, E. M.(1897)
111 E. Church St., Marshalltown, Ia., OAL
SISSON, RAYMOND J.(1928)
753 David Whitney Bldg., Detroit, Mich., Op.
*SKILLERN, R. H.(1910).1811 Spruce St., Philadelphia, Pa., R
*SKILLERN, S. R., JR.(1925)
1734 Pine St., Philadelphia, Pa., ALR
SKIRBALL, JOSEPH J.(1927)
353 Commonwealth Ave., Boston, Mass., OALR
SLEIGHT, R. D.(1906).303 Post Bldg., Battle Creek, Mich., Op.
SLOAN, HENRY L.(1919)....6 W. 7th St., Charlotte, N. C., Op.
SLOCUM, GEO.(1906)..311 S. State St., Ann Arbor, Mich., Op.
*SMIT, WM. MAURICE(1928)
719 Missouri Theatre Bldg., St. Louis, Mo., ALR
SMITH, ARTHUR E., Donaldson Bldg., Minneapolis, Minn., OALR
*SMITH, CARLIE SOUTER(1927)
214 N. Jefferson Ave., Springfield, Mo., OALR

SMITH, DORLAND (1910)
834 Myrtle Ave., Bridgeport, Conn., Op.A
*SMITH, D. S. (1917) . . . . State Bank Bldg., La Crosse, Wis., OALR
SMITH, E. TERRY . . . . . 36 Pearl St., Hartford, Conn., Op.
SMITH, GEO. B. (1914) . . . . . Harlin Hospital, Rome, Ga., OALR
SMITH, H. A. (1925)
Colo. Bank & Trust Bldg., Delta, Colo., OALR
SMITH, J. M . . . . . 134½ N. Patterson St., Valdosta, Ga., OALR
SMITH, R. C . . . . . Board of Trade Bldg., Superior, Wis., OALR
SMITH, S. McC . . . . . 1429 Spruce St., Philadelphia, Pa., ALR
SMITH, STANLEY . . . . . 121 University Pl., Pittsburgh, Pa., Op.
SMITH, VICTOR C. (1912)
1707 Pere Marquette Bldg., New Orleans, La., Op.
*SNAPP, CARL FOSTER (1928)
604 Med. Arts Bldg., Grand Rapids, Mich., ALR
SNEED, CARL M . . . . . 506 W. Broadway, Columbia, Mo., OALR
SNELL, ALBERT C. (1925)
53 S. Fitzhugh St., Rochester, N. Y., Op.
SNYDER, W. H . . . . . 220 Michigan St., Toledo, O., OALR
SOMBERG, J. S. (1923) . . . . 60 E. 67th St., New York, N. Y., Op.
*SONNENSCHEIN, R. (1915)
180 N. Michigan Ave., Chicago, Ill., ALR
*SPAETH, EDMUND B. (1928)
N. E. Cor. 20th and Chestnut Sts., Philadelphia, Pa., Op.
SPAKE, LAVERNE B. (1920)
320 Brotherhood Bldg., Kansas City, Kans., ALR
SPALDING, J. A. (1896) . . . . 627 Congress St., Portland, Me., Op.A
*SPENCER, FRANK R. (1904)
Physicians Bldg., Boulder, Colo., OALR
SPENCER, GEO. ALBERT (1928)
1005 Seventh St., Sacramento, Cal., OALR
SPENCER, W. H. . 1830 S. Rittenhouse Sq., Philadelphia, Pa., ALR
SPENGLER, JOHN ARTHUR (1916)
423 S. Main St., Geneva, N. Y., Op.
*SPITZE, E. C. (1916) . . . . Murphy Bldg., East St. Louis, Ill., Op.
SPROULE, RALPH P. (1928)
141 E. Wisconsin Ave., Milwaukee, Wis., OALR
STAHLMAN, FRED C. . Commercial Bldg., Waynesburg, Pa., OALR
STANBERRY, HENRY . . . . . 556 Doctors Bldg., Cincinnati, O., Op.
*STANFORD, J. B . . . . . 1052 Madison Ave., Memphis, Tenn., OALR
*STANTON, HARMON LAMONTTE (1928)
Cleveland Life Bldg., Evansville, Ind., OALR
STARK, JESSE B. (1925)
45 Park Ave., New York City, N. Y., Op.
STARK, WM. B. (1927) . . . . Mayo Clinic, Rochester, Minn., ALR
*STAUFFER, FRED (1912)
Medical Arts Bldg., Salt Lake City, Utah, OALR
*STEELE, WILLARD (1928)
Provident Bldg., Chattanooga, Tenn., OALR
STEGMAN, LOUIE V. (1919) Sanitarium, Battle Creek, Mich., Op.

STEIM, J. M.(1914)
First Natl. Bank Bldg., New Kensington, Pa., Op.
STEIN, O. J.....30 N. Michigan Blvd., Chicago, Ill., ALR
STEINFELD, A. L.(1922).....125 15th St., Toledo, O., OALR
STEVENSON, D. W.....165 E. Market St., Akron, O., OALR
*STEVENSON, WALTER(1915)..W. C. U. Bldg., Quincy, Ill., OALR
STEWART, WALTER E.(1920)
Tribune Bldg., Terre Haute, Ind., OALR
STIEREN, E.(1905).626 Union Trust Bldg., Pittsburgh, Pa., Op.
STILES, CHAS. M.(1908)
1130 Spruce St., Philadelphia, Pa., Op.
STILLSON, H.....Seaboard Bldg., Seattle, Wash., OALR
STIMSON, GEORGE W.(1919)...Packard Apts., Warren, O., OALR
STINSON, WM. DAVID(1928)
805 Med. Arts Bldg., Memphis, Tenn., ALR
STITT, H. L.(1927).....656 Doctors Bldg., Cincinnati, O., ALR
STOCKLER, J. A.(1917)..332 North Fifth St., Reading, Pa., OALR
STOKES, WM. H.(1926)...4105 Live Oak St., Dallas, Tex., Op.
STOTTER, ARTHUR L.(1916)
1148 Euclid Ave., Cleveland, O., OALR
STOUT, PHILIP S....Medical Arts Bldg., Philadelphia, Pa., ALR
STOUTENBOROUGH, WM. A.(1927)
318 East State St., Columbus, O., OALR
STRADER, GEO. L.(1904)
408 Hynds Bldg., Cheyenne, Wyo., OALR
*STRAUSS, J. F.(1925)..104 S. Michigan Blvd., Chicago, Ill., ALR
STRICKLER, D. A.....Republic Bldg., Denver, Colo., Op.
STRONG, J. C.(1915)...810 Harrison Ave., Leadville, Colo., OALR
STROUD, EDWARD FRANK(1926)
408 City Natl. Bank Bldg., Corpus Christi, Tex., ALR
STROUSE, FREDERICK M.(1920)
1301 Spruce St., Philadelphia, Pa., ALR
STROUT, E. S.(1907)...74 S. 11th St., Minneapolis, Minn., OALR
STROUT, G. E.(1921).Donaldson Bldg., Minneapolis, Minn., OALR
*STUCKY, J. A.(1896)...Fayette Bk. Bldg., Lexington, Ky., OALR
*STUEBER, F. G.(1905).....209 W. North St., Lima, O., OALR
STUEBER, PAUL J.(1917).....209 W. North St., Lima, O., OALR
STURGES, LEIGH F.(1920)
121 E. 60th St., New York City, N. Y., ALR
STURM, S. A.(1916)
7036 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*SUKER, GEO. F.(1896).25 E. Washington St., Chicago, Ill., Op.
SULLIVAN, J. J., JR.(1923)
228 Scranton Life Bldg., Scranton, Pa., ALR
SULZER, G. A.(1907).....327 E. State St., Columbus, O., OALR
*SULZMAN, FRANK MALCOLM(1922)
1831 Fifth Ave., Troy, N. Y., OALR
*SUMMERS, WM. S.(1927)
1613 David Whitney Bldg., Detroit, Mich., OALR

SWAB, CHARLES MARION (1928)
Medical Arts Bldg., Omaha, Neb., Op.
SWAN, C. J. (1911) .....636 Church St., Evanston, Ill., OALR
SWEET, ROBERT B. (1926)
Pacific S. W. Bk. Bldg., Long Beach, Cal., OALR
SWERDFEGER, E. B. (1927)
1008 Republic Bldg., Denver, Colo., ALR
SWETNAM, C. K. (1915) . . Masonic Temple, Prescott, Ariz., ALR

T

TANGEMAN, H. F. (1919)
606 Union Central Bldg., Cincinnati, O., ALR
TAQUINO, GEORGE J. (1920)
208 Audubon Bldg., New Orleans, La., ALR
TARUN, WILLIAM .....605 Park Ave., Baltimore, Md., OALR
*TAYLOR, CHARLES B. (1921) .....Ottumwa, Ia., ALR
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25 E. Washington St., Chicago, Ill., ALR
TAYLOR, M. C. (1915) ....1204 May Bldg., Pittsburgh, Pa., Op.
*TEALL, RAYMOND EDWIN (1928)
1100 Rialto Bldg., Kansas City, Mo., ALR
*THIGPEN, C. A. .....401 So. Court St., Montgomery, Ala., OALR
*THOMAS, C. D. (1914)
Central Natl. Bank Bldg., Peoria, Ill., OALR
THOMAS, FRANCIS W. (1916)
327 E. State St., Columbus, O., OALR
THOMAS, H. C. (1926) .....Franklin Bldg., Oakland, Cal., OALR
THOMAS, JAMES W. (1928)
726 Sec. Mutual Bldg., Lincoln, Neb., OALR
*THOMASON, H. E. .....Rialto Bldg., Kansas City, Mo., ALR
THOMASSON, W. J. (1910) ....942 York St., Newport, Ky., ALR
THOMPSON, E. H .....8 W. 9th St., Cincinnati, O., OALR
THOMPSON, G. H. (1909)
18 Ashland St., North Adams, Mass., OALR
THOMPSON, HOWARD E. (1928)
1100 Main St., Dubuque, Ia., OALR
THOMPSON, PETER H. (1914)
308 Commonwealth Ave., Boston, Mass., Op.
THOMSON, J. J. (1907)
Esplanade Gardens, East Lincoln Ave., Mt. Vernon, N. Y., ALR
*TILDERQUIST, D. L. (1925)
425 Bradley Bldg., Duluth, Minn., OALR
TIMBERMAN, ANDREW .....318 E. State St., Columbus, O., OALR
TINDOLPH, L. W. .....1st Natl. Bank Bldg., Olean, N. Y., ALR
TINGLEY, LOUISA PAINE (1913)
9 Massachusetts Ave., Boston, Mass., Op.
*TITSWORTH, GUY .....111 W. 4th St., Sedalia, Mo., OALR
*TIVNEN, RICHARD J ....30 N. Michigan Ave., Chicago, Ill., OALR
*TOBEY, GEO. L., JR. 270 Commonwealth Ave., Boston, Mass., ALR

*TODD, GROVER CLEVELAND (1922)
307 Westinghouse Bldg., Pittsburgh, Pa., ALR
*TOMASSENE, R. A. (1922)
Wheeling Bank & Trust Bldg., Wheeling, W. Va., OALR
TOMLIN, W. S. (1912)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
TOMS, S. W. S. (1909) ....120 S. Broadway, Nyack, N. Y., OALR
*TOOKER, C. W. (1907) .....Carleton Bldg., St. Louis, Mo., Op.
TOWNSEND, JOHN FERRARS (1923)
85 Wentworth, Cor. St. Phillips St., Charleston, S. C., OALR
*TRIBLE, G. B. (1920)
1801 Eye St., N. W., Washington, D. C., ALR
TRIMBLE, CLARENCE S. (1920)
405 Citizens Natl. Bank Bldg., Emporia, Kan., OALR
TRIPP, IRA A. (1920) .....436 Rose Bldg., Cleveland, O., OALR
TRIPP, LEROY R. .....307 Security Bldg., Sioux City, Ia., OALR
*TUCKER, GABRIEL (1923)
Graduate Hospital, 19th & Lombard St., Philadelphia, Pa., B
*TUCKER, JAMES C. (1920) ..114 So. 6th St., Beatrice, Neb., OALR
TUCKERMAN, W. C. (1911) 733 Osborn Bldg., Cleveland,O., Op.
TUCKERMAN, W. H. .....733 Osborn Bldg., Cleveland,O., OALR
TURNBULL, F. M. (1916)
806 Pacific Mutual Bldg., Los Angeles, Cal., ALR
*TURNER, H. H. (1914)
902 Professional Bldg., Pittsburgh, Pa., Op.
*TURNER, OLIVER W. (1909) Masonic Temple, Augusta, Me., OALR

U

UPHAM, HELEN F. (1907)
305 Third Ave., Asbury Park, N. J., OALR
UREN, C. T. (1919)
537 City Natl. Bank Bldg., Omaha, Neb., ALR
URNER, M. H. ...2600 Union Central Bldg., Cincinnati, O., OALR

V

VAIL, DERRICK T. .....24 E. 8th St., Cincinnati, O., Op.
*VAIL, H. H. (1924) .....24 E. 8th St., Cincinnati, O., ALR
*VAN KIRK, FRANK J. (1920)
Bellingham Natl. Bank Bldg., Bellingham, Wash., OALR
VANKIRK, V.E. ...626 Union Trust Bldg., Pittsburgh, Pa., Op.
VAN METER, EUGENE R. (1927)
4473 Washington Ave., St. Louis, Mo., ALR
VAN POOLE, G. M. (1925)
Young Bldg., Honolulu, T. Hawaii, OALR
*VAUGHAN, G. E. .....711 3rd St., Louisville, Ky., OALR
VAUGHN, E. H. .....Citizens Natl. Bank, Tyler, Tex., OALR
VEASEY, C. A. (1905) .....Paulsen Bldg., Spokane, Wash., OALR
VEASEY, C. A., JR. (1927)
404 Paulsen Bldg., Spokane, Wash., OALR

VINSONHALER, F. (1902)
Urquhart Bldg., Little Rock, Ark., OALR
*VOIGT, C. B. (1912) ..... 1702 Broadway, Mattoon, Ill., OALR
VON COLDITZ, G. THOMSEN
25 E. Washington St., Chicago, Ill., OALR
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25 E. Washington St., Chicago, Ill., Op.

W

*WAGENER, HENRY P. (1926) Mayo Clinic, Rochester, Minn., Op.
WAGERS, ARTHUR J. (1920)
The Lenox, 13th and Spruce Sts., Philadelphia, Pa., ALR
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1003 Pere Marquette Bldg., New Orleans, La., ALR
WALKER, CHAS. C. (1920)
604 Maccabee Bldg., Detroit, Mich., Op.
WALKER, C. E. (1920) .. 517 Jacobson Bldg., Denver, Colo., Op.
*WALKER, GEO. H. (1927) .... 172 Main St., Winona, Minn., OALR
WALKER, HERBERT S. ... 30 N. Michigan Ave., Chicago, Ill., OALR
WALKER, WALDO WEBSTER (1928)
270 Commonwealth Ave., Boston, Mass., ALR
WALSH, JOSEPH MARK (1922) ..... Rapid City, S. D., OALR
WALTER, WILL ..... 1507 Hinman Ave., Evanston, Ill., OALR
WARREN, WILLIAM C., JR. (1928)
618 Atlanta Natl. Bank Bldg., Atlanta, Ga., ALR
WARWICK, HAROLD LYNWOOD (1928)
913 Med. Arts Bldg., Fort Worth, Tex., ALR
WATKINS, EDWIN DIAL (1926)
1412 Exchange Bldg., Memphis, Tenn., OALR
WATSON, H. D. (1915) .. 151 Front St., Binghamton, N. Y., OALR
WATSON, JAMES A. ..... 74 S. 11th St., Minneapolis, Minn.,
*WATSON, R. S. (1923) .... Graebrier Bldg., Saginaw, Mich., OALR
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Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., OALR
*WEAVER, T. W. (1914) ..... 201 Main St., Wichita, Kan., Op.
WEBER, HENRY CHARLES (1928)
Naval Hospital, Great Lakes, Ill., OALR
*WEED, HARRY M. (1908) . 196 Linwood Ave., Buffalo, N. Y., Op.
WEEKS, JOHN E. ..... Dunthorpe Rt. 8, Portland, Ore., Op.
WEEKS, WEBB WILLIAM (1923)
20 E. 53rd St., New York City, N. Y., Op.
*WEIH, E. P. (1920) ..... Wilson Bldg., Clinton, Ia., OALR
WEIL, A. I. ..... 717 Audubon Bldg., New Orleans, La., ALR
WEIMER, EDGAR S. ..... Highland Bldg., Pittsburgh, Pa., Op.
WEINBERGER, NELSON S. (1928)
Robert Packer Hospital, Sayre, Pa., OALR

WEINSTEIN, M. A.(1927)
1309 Spruce St., Philadelphia, Pa., ALR
WEISSER, E. A.....806 May Bldg., Pittsburgh, Pa., Op.
*WELCH, ROBERT S. G.(1921)
46 State St. Circle, Annapolis, Md., OALR
WELLS, DAVID W.(1908)
Hotel Westminster, Boston, Mass., Op.
WELLS, GEO. S.....8 E. Arrellaga, Santa Barbara, Cal., ALR
WELTY, C. F.(1926).....210 Post St., San Francisco, Cal., ALR
WENCKE, CARL G.,
Battle Creek Sanit., Battle Creek, Mich., ALR
*WENDEL, J. S.(1922)
744 David Whitney Bldg., Detroit, Mich., ALR
*WESTLAKE, SAMUEL B.
Beaumont Med. Bldg., St. Louis, Mo., ALR
WETHERBEE, CHARLES (1926).....Twin Falls, Ida., OALR
WHALEN, EDWARD JOSEPH (1928)
780 Main St., Hartford, Conn., ALR
WHEELER, ERNEST C.....Rust Bldg., Tacoma, Wash., OALR
*WHEELER, J. M.(1920)
30 W. 59th St., New York City, N. Y., Op.
WHEELER, MERRITT W.1027 Lowry Bldg., St. Paul, Minn., OALR
WHELAN, G. L.(1925)..257 S. 16th St., Philadelphia, Pa., ALR
*WHERRY, W. P.(1910)
1500 Medical Arts Bldg., Omaha, Neb., OALR
WHISNANT, A. M.517 Professional Bldg., Charlotte, N. C., OALR
*WHITAKER, J.(1917).....905 Congress Bldg., Miami, Fla., OALR
WHITE, CHAS. P., 507 Citizens Bk. Bldg., Wilmington, Del.,
WHITE, E. H.(1912)
756 Sherbrooke St., W., Montreal, Can., ALR
WHITE, WILLIAM BEVERLY(1928)
813 Gurley Bldg., Stamford, Conn., OALR
WIENER, ALFRED(1909)
550 Park Ave., New York City, N. Y., OALR
*WIENER, MEYER.....900 Carleton Bldg., St. Louis, Mo., Op.
WILBUR, EDWARD P.,
403 Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
*WILDER, WM. H.(1896)
122 S. Michigan Blvd., Chicago, Ill., Op.
WILLARD, L. M.(1915).....520 3rd St., Wausau, Wis., OALR
WILLIAMS, HORACE J.(1922)
5908 Green St., Germantown, Philadelphia, Pa., ALR
*WILLIAMS, M. M.(1927)..148 Adams Ave., Scranton, Pa., Op.
*WILLIAMS, W. C.(1906).....Jefferson Bldg., Peoria, Ill., OALR
*WILLIAMS, WALTON H.(1921)
Castell Bldg., Middletown, O., OALR
WILLSON, WESLEY W.(1928)
563 David Whitney Bldg., Detroit, Mich., OALR
*WINDHAM, ROBERT EDWARD(1928).....Longview, Wash., OALR

*WINGER, IRA B.(1927).....225 Michigan St., Toledo, O., OALR
*WINTER, GEORGE E.(1921)..44 Sun Bldg., Jackson, Mich., OALR
WINTER, JOHN A.(1923)..205 W. 2nd St., Duluth, Minn., OALR
*WISE, RALPH C.(1914).....Mansfield, O., OALR
*WOLD, K. C.(1924).1027 Med. Arts Bldg., St. Paul, Minn., OALR
*WOLFE, CLAUDE T.(1916).Heyburn Bldg., Louisville, Ky., OALR
*WOLFE, OTIS R.(1919)
415 Masonic Temple, Marshalltown, Ia., OALR
WOOD, DOUGLAS.....Donaldson Bldg., Minneapolis, Minn., OALR
WOOD, EARL LEROY(1928)
33 Lincoln Park, Newark, N. J., ALR
*WOOD, V. VISSCHER(1928)
201 Humboldt Bldg., St. Louis, Mo., ALR
*WOODRUFF, FREDERICK E.,
Metropolitan Bldg., St. Louis, Mo., Op.
*WOODRUFF, H. W.....Morris Bldg., Joliet, Ill., OALR
WOODRUFF, T. A.....Mercer Bldg., New London, Conn., Op.
WOODRUFF, WALTER STUART(1927)
421 Huguenot St., New Rochelle, N. Y., ALR
WOODS, R. H.....Neustadt Bldg., La Salle, Ill., Op.
WOODSON, BURBANK PALMER(1928).....Temple, Tex., OALR
*WOODSON, J. M.(1914)
Cor. Avenue G and Fifth, Temple, Tex., OALR
WOODWORTH, WM. P.(1927)
1424 Maccabees Bldg., Detroit, Mich., OALR
*WORMLEY, H. R.....Talcott Bldg., Rockford, Ill., OALR
WRIGHT, C. DARCY(1921)
307 Yeates Bldg., Minneapolis, Minn., OALR
WRIGHT, J. B.....Professional Bldg., Raleigh, N. C., ALR
WRIGHT, J. W.....Central Natl. Bldg., Columbus, O., Op.
*WRIGHT, J. W. M..Hume-Mansur Bldg., Indianapolis, Ind., ALR
WRIGHT, WALTER WALKER(1926)
143 College St., Toronto, Can., Op.
WURDEMANN, HARRY V.
320 Stimson Bldg., Seattle, Wash., Op.
*WURTZ, W. J. M.(1913) 528 Delaware Ave., Buffalo, N. Y., ALR
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Y

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YAZEL, HERMAN E.....733 Rialto Bldg., Kansas City, Mo., OALR
YOST, WALTER MILTON(1928)
Peoples Bk. Bldg., Rochester, Pa., Op.
*YOUNG, CHARLES A.(1926)
Shenandoah Life Bldg., Roanoke, Va., Op.
*YOUNG, H. B.(1897)..204 Jefferson St., Burlington, Iowa, OALR

Z

ZEHNDER, A. CHARLES (1919)
188 Roseville Ave., Newark, N. J., Op.
*ZEMER, S. G. . . . . 726 Security Mutual Bldg., Lincoln, Neb., OALR
*ZENTMAYER, WILLIAM . . 1506 Spruce St., Philadelphia, Pa., Op.
ZINKMAN, A. M. (1920)
The Rochambeau, Washington, D. C., ALR
*ZUBAK, MATTHEW FRANCIS . 75 12th St., Wheeling, W. Va., OALR

GEOGRAPHIC LIST

ALABAMA

BIRMINGHAM

HARRISON, W. G. ..... Empire Bldg., OALR
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SCHWARTZ, WM. A. ..... OALR

PRESCOTT

SWETNAM, C. K. ..... Masonic Temple, ALR

ARKANSAS

FORT SMITH

BUCKLEY, J. H. ..... 1st National Bank Bldg., OALR
DORENTE, D. R. ..... 1st National Bank Bldg., OALR
MOULTON, EVERETT C. ..... Merchants National Bank Bldg., OALR
MOULTON, HERBERT ..... Merchants National Bank Bldg., OALR

HOT SPRINGS

KING, O. H. ..... Dugan-Stuart Bldg., OALR
ROWLAND, JOHN F. ..... Thompson Bldg., OALR
SHORT, Z. N. ..... Dugan-Stuart Bldg., OALR

LITTLE ROCK

CALDWELL, ROBERT ..... 831 New Doneghey Bldg., OALR
MAHONEY, PAUL L. ..... Doneghey Bldg., ALR
VINSONHALER, F. ..... Urquhart Bldg., OALR

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DAYTON, G. O.....245 Bradbury Bldg., OALR
DETLING, FRANK E.....500 S. Lucas St., OALR
FISHER, CARL.....1930 Wilshire Blvd., Op.
GOSNEY, C. W.....1009 Haas Bldg., Op.
IRVINE, A. R.....1144 Roosevelt Bldg., Op.
KIEFER, HUGO A.....Brockman Bldg., OALR
KRESS, GEO. H.....245 Bradbury Bldg., OALR
LEFLER, ANNA B.....605 S. Broadway, OALR
LEWIS, EUGENE R.....Roosevelt Bldg., ALR
MAGHY, CHAS. A.....1136 W. 6th S., Op.
TURNBULL, F. M.....806 Pacific Mut. Bldg., ALR

OAKLAND

O'CONNOR, RODERIC P.....1904 Franklin St., Op.
THOMAS, H. C.....Franklin Bldg., OALR

PASADENA

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DURR, S. A.....1304 Medico-Dental Bldg., Op.
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BARKAN, ADOLPH.....Union Trust Bldg., OALR
BARKAN, HANS.....490 Post St., Op.

BLAKE, WM. F.....490 Post St., Op.
BROWN, H. ALEXANDER.....291 Geary St., Op.
DERIVER, J. P.....Veterans Bureau, OALR
DOLMAN, PERCIVAL.....490 Post St., Op.
GRAHAM, H. B.....490 Post St., ALR
GREEN, A. S.....The Green's Eye Hospital, Op.
GREEN, L. D.....The Green's Eye Hospital, Op.
HEBERT, ARTHUR W.....Flood Bldg.
SELFRIDGE, G. L.....384 Post St., ALR
SEWALL, E. C.....Stanford Univ. Hospital, ALR
WELTY, C. F.....210 Post St., ALR

SANTA BARBARA

JEAN, G. W.....7 E. Valerio, Op.
LEWIS, J. D.....1534 Estado St., OALR
MELLINGER, WM. J.....120 E. Sola St., ALR
WELLS, GEO. S.....8 E. Arrellaga, ALR

SANTA MONICA

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LEVINGOOD, H. W.....327 Wilshire Blvd., OALR
SHERMAN, MAJ. A. ED..M.C., U. S. A. Headq., Clover Field, OALR

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ALAMOSA

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SPENCER, FRANK R.....Physicians Bldg., OALR

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DENNIS, F. L.....301 Ferguson Bldg., ALR
KETTELKAMP, F. O.....301 Ferguson Bldg., ALR
NEEPER, EDW. R.....Exchange Natl. Bank Bldg., Op.
PATTERSON, JAS. A.....Burns Bldg., OALR

DELTA

SMITH, H. A.....Colo. Bk. and Trust Bldg., ALR

DENVER

ALLEN, J. H.....Republic Bldg., ALR
BANE, W. C.....1005 Republic Bldg., OALR
BANE, WM. M.....1005 Republic Bldg., OALR
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HASPEL, M. DAVID ..... 603 Maison Blanche Bldg., ALR
LANDFRIED, C. J ..... 607 Baronne Bldg., ALR
LEJEUNE, F. E ..... 632 Maison Blanche Bldg., ALR
LYNCH, R. C ..... Maison Blanche Bldg., ALR
SMITH, VICTOR C ..... 1707 Pere Marquette Bldg., Op.
TAQUINO, GEORGE J ..... 208 Audubon Bldg., Op.
WAGNER, W. A ..... 1003 Pere Marquette Bldg., ALR
WEIL, A. I ..... 717 Audubon Bldg., ALR

SHREVEPORT

LARUE, C. L ..... Highland Clinic, OALR
MOISE, ALLYN B ..... Med. Arts Bldg., ALR
SCALES, J. L ..... Commercial Natl. Bk. Bldg., OALR

MAINE

AUGUSTA

TURNER, OLIVER W.....Masonic Temple, OALR

PORTLAND

BEACH, S. J.....704 Congress St., Op.
GILBERT FRANK Y.....148 Park St., OALR
GORDON, CHAS. H.....46 Deering St., ALR
HASKELL, ALFRED W.....129 Park St., Op.
HOLT, E. E. JR.....723 Congress St., Op.
O'SULLIVAN, T. J.....10 Pine St., ALR
SPALDING, J. A.....627 Congress St., Op.A

WATERVILLE

HILL, FREDERICK T.....Professional Bldg., ALR
HILL, HOWARD F.....Professional Bldg., Op.

MARYLAND

ANNAPOLIS

WELCH, ROBERT S. G.....46 State St. Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR.....330 N. Charles St., OALR
FRIEDENWALD, HARRY.....1212 Eutaw Pl., Op.A
FRIEDENWALD, J. S.....1212 Eutaw Pl., Op.
GOLDBACK, L. J.....1012 N. Charles St., Op.
KEMLER, JOS. J.....1908 Eutaw Pl., OALR
LOOPER, EDWARD ANDERSON.....605 Park Ave., OALR
PETERMAN, H. E.....512 Cathedral St., OALR
SAVAGE, M. M.....1720 Eutaw Pl., OALR
TARUN, WILLIAM.....605 Park Ave., OALR
WOODS, HIRAM.....842 Park Ave., Op.A

CUMBERLAND

JONES, E. L.....First National Bank Bldg., OALR
ROBINSON, H. T.....Medical Bldg., OALR
SHARRETT, GEO. O.....119 Bedford, OALR

MASSACHUSETTS

BOSTON

BRYANT, ALICE G.....502 Beacon St., ALR
CARVILLE, L. MAUD.....82 Commonwealth Ave., Op.
CHENERY, W. E.....377 Commonwealth Ave., ALR
CORBETT, J. J.....520 Beacon St., OALR
DERBY, GEO. S.....23 Bay State Road, Op.
FREEDMAN, L. M.....475 Commonwealth Ave., ALR
GREENWOOD, A.....82 Commonwealth Ave., Op.
HURLEY, E. D.....419 Boylston St., Op.
INGLIS, HARRY J.....43 Bay State Rd., ALR
LANCASTER, W. B.....529 Commonwealth Ave., Op.

LEWIS, J. P.....543 Boylston St., ALR
MOSHER, H. P.....828 Beacon St., ALR
QUINCY, J. E.....270 Commonwealth Ave., ALR
RICE, G. B.....270 Commonwealth Ave., ALR
RICHARDS, L. G.....270 Commonwealth Ave., ALR
ROWLAND, W. D.....220 Clarendon St., Op.
SCHALL, L. A.....520 Commonwealth Ave., ALR
SKIRBALL, J. J.....353 Commonwealth Ave., OALR
STANDISH, MYLES.....51 Hereford St., Back Bay, Op.
THOMPSON, PETER H.....308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE.....9 Massachusetts Ave., Op.
TOBEY, GEO. L., JR.....270 Commonwealth Ave., ALR
WALKER, WALDO W.....270 Commonwealth Ave., ALR
WELLS, DAVID W.....Hotel Westminister, Op.

CAMBRIDGE

HOWE, LUCIEN.....14 Concord St., Op.

CLINTON

MONAHAN, J. A.....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R.....151 Rock St., ALR

FRAMINGHAM

JESSAMAN, L. W.....132 Union Ave., OALR

GARDNER

BONE, HERMAN D.....19 Pleasant St., OALR

HOLYOKE

BURKE, JOHN R.....207 Elm St., OALR
HUSSEY, E. J.....245 Maple St., OALR

LAWRENCE

CONLON, F. A.....301 Essex St., OALR
MERRILL, W. HOWE.....301 Essex St., OALR

LYNN

JONES, E. W.....44 Atlanta St., Op.A
O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....68 Walnut St., OALR

NORTH ADAMS

THOMPSON, G. H.....18 Ashland St., OALR

NORTHAMPTON

COLLINS, J. D.....150 Main St., ALR
DOW, FRANK E.....4 West St., Op.

PALMER

MOORE, G. A.....Main St., OALR

SALEM

HENNESSEY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....67 Chestnut St., OALR
CARLETON, RALPH.....10 Temple St., Op.
GALLAGHER, J. H. C.....1537 Main St., ALR
IRWIN, VINCENT J., JR.....1634 Main St., OALR

TAUNTON

BAKER, HARRY B.....59 Main St., OALR

WORCESTER

CAHILL, J. W.....390 Main St., OALR
CROSS, A. E.....390 Main St., Op.
ESTABROOK, CHAS. T.....857 Pleasant St., OALR
MESSIER, A. E.....390 Main St., OALR
RICE, JOHN E.....390 Main St., OALR

MICHIGAN

ANN ARBOR

FURSTENBERG, A. C.....330 S. State St., ALR
SLOCUM, GEO.....311 S. State St., Op.

BATTLE CREEK

COLVER, B. N.....Sanitarium, ALR
FRASER, R. H.....Battle Creek Sanitarium, ALR
HAUGHEY, WILFRID.....303 Post Bldg., OALR
SLEIGHT, R. D.....303 Post Bldg., Op.
STEGMAN, LOUIE V.....Sanitarium, Op.
WENCKE, CARL G.....Battle Creek Sanitarium, ALR

BAY CITY

BAKER, CHAS. H.....Crapo Block, OALR

DETROIT

AMBERG, EMIL.....622 Maccabee Bldg., ALR
BEATTIE, ROBERT.....David Whitney Bldg., OALR
BEGLE, HOWELL L.....1513 Longfellow Ave., Op.
BENTLEY, NEIL.....1161 David Whitney Bldg., OALR
BERNSTEIN, E. J.....10 Peterboro, OALR
BROWN, A. O.....742 Maccabee Bldg., OALR
CAMPBELL, D. A.....David Whitney Bldg., OALR
CAMPBELL, DON M.....David Whitney Bldg., OALR
CAMPBELL, M. D.....David Whitney Bldg., OALR
CLIFFORD, T. P.....1320 David Whitney Bldg., OALR
CONNOR, RAY.....1410 Stroh Bldg., OALR
DEFNET, WM. A.....1309 Stroh Bldg., ALR
FOWLER, WM.....1048 Maccabee Bldg., OALR
GLOWACKI, BEN F.....Maccabee Bldg., OALR
GOUX, RAYMOND SALOT.....545 David Whitney Bldg., OALR

GRANT, LEE E.....David Whitney Bldg., OALR
HALE, ARTHUR S.....3790 Woodward Ave., OALR
HARRELL, VOSS.....Hofman Bldg., ALR
HEATH, PARKER.....David Whitney Bldg., Op.
HUGHES, RAY W.....1501 David Whitney Bldg., OALR
KEATING, T. E.....5050 Woodward Ave., OALR
MCCLELLAND, CARL C.....1857 David Whitney Bldg., OALR
MAIRE, LEWIS E.....710 Kresge Bldg., OALR
MERCER, R. E.....David Whitney Bldg., LR
MERKEL, CHARLES C.....1409 David Whitney Bldg., ALR
MINER, S. G.....2641 E. Jefferson Ave., ALR
MORTON, J. B.....90 E. Warren Ave., OALR
PARKER, WALTER R.....David Whitney Bldg., Op.
PEIRCE, HORACE W.....704 Medical Arts Bldg., ALR
POOS, E. E.....13300 Woodward Ave., OALR
RENAUD, G. L.....510 Joseph Campan Ave., OALR
RYERSON, F. L.....Fine Arts Bldg., OALR
SANDERSON, H. H.....David Whitney Bldg., OALR
SHURLY, BURT R.....62 Adams Ave., W., ALR
SIMPSON, H. LEE.....1409 David Whitney, Bldg., ALR
SISSON, RAYMOND J.....753 David Whitney Bldg., Op.
SUMMERS, WM. S.....1613 David Whitney Bldg., OALR
WALKER, CHAS. C.....604 Maccabee Bldg., Op.
WENDEL, J. S.....744 David Whitney Bldg., ALR
WILLSON, W. W.....563 David Whitney Bldg., OALR
WOODWORTH, WM. P.....1424 Maccabee Bldg., OALR

ESCANABA

BOYCE, W. B.....1107 Ludington St., OALR

GRAND RAPIDS

DEAN, ALFRED.....308 Med. Arts Bldg., Op.
GRANT, P. T.....1407 Grand Rapids Natl. Bk. Bldg., OALR
ROBERTSON, F. D.....Ashton Bldg., OALR
ROLLER, LOUIS A.....446 Paris Ave., S. E., Retired
SNAPP, CARL F.....Med. Arts Bldg., ALR

IONIA

SEDGWICK, O. W.....OALR

IRON MOUNTAIN

BOYCE, G. H.....1st Natl. Bk. Bldg. OALR

JACKSON

WINTER, GEORGE E.....44 Sun Bldg., OALR

KALAMAZOO

FULKERSON, C. B.....308 Kalamazoo National Bank Bldg., OALR
GRANT, FRED E.....Kalamazoo National Bank Bldg., OALR
WILBUR, EDWARD P...403 Kalamazoo National Bank Bldg., OALR

LANSING

BURDICK, AUSTIN F.....416 Tussing Bldg., OLAR
ELLIS, CHARLES W.....ALR

MENOMINEE

ELWOOD, CALVIN R.....Spies Bldg., OALR
JONES, WM. S.....522 Sheridan Rd., OALR

MT. CLEMENS

CROMAN, JOS. M., JR.....Washington Apts., OALR

ST JOSEPH

MCDERMOTT, JOHN J.....1871 Niles Ave., OALR

SAGINAW

MCKINNEY, A. R.....704 2nd Natl. Bk. Bldg., OALR
ROGERS, A. S.....105 S. Jefferson Ave., OALR
WATSON, R. S.....Graebrier Bldg., OALR

TRAVERSE CITY

HOLDSWORTH, FRANK.....Peoples Bk. Bldg., OALR

MINNESOTA

ALBERT LEA

FOLKEN, FRANK G.....306 Albert Lea State Bk. Bldg., OALR

DULUTH

BRIGGS, F. W.....Fidelity Bldg., OALR
KNAPP, F. N.....329 Bradley Bldg., OALR
SHASTID, T. H.....10th Ave. Med. Bldg., Op.
TILDERQUIST, D. L.....425 Bradley Bldg., OALR
WINTER, JOHN A.....205 W. 2nd St., OALR

FERGUS FALLS

KITTLESON, THEODORE..... OALR

HIBBING

MORSMAN, L. W.....205 Power Bldg., OALR
RUBY, F. M..... OALR
SINAMARK, ANDREW.....205 Power Bldg., OALR

MANKATO

JAMES, J. H.....National City Bank Bldg., OALR

MINNEAPOLIS

BROWN, EDWARD J.....900 Nicollet Ave., OALR
CAMP, W. E.....716 Donaldson Bldg., OALR
CAMPBELL, ROBERT A.....520 La Salle Bldg., OALR
CLARK, H. S.....La Salle Bldg., OALR
DORGE, RICHARD IRVING.....Silvertsen Clinic, OALR
FINK, WALTER H.....426 La Salle Bldg., OALR
HANSEN, ERLING W.....910 Donaldson Bldg., OALR
HOWARD, W. H.....1009 Nicollet Ave., OALR
LITCHFIELD, J. T.....609 Yeats Bldg., OALR
LOOMIS, E. A.....Donaldson Bldg., OALR
MACNIE, J. S.....74 S. 11th St., OALR
MATTHEWS, JUSTUS.....1021 Metropolitan Bldg., LR
MORTON, HOWARD MCI.....418 Insurance Bldg., Op.

NEWHART, HORACE.....Donaldson Bldg., ALR
OBERG, C. M.....Syndicate Bldg., OALR
PATTERSON, W. E.....74 S. 11th St., OALR
PHELPS, KENNETH A.....730 La Salle Bldg., OALR
PRATT, FRED J.....Metropolitan Bk. Bldg., OALR
PRATT, JOHN A.....Metropolitan Bk. Bldg., OALR
REYNOLDS, J. S.....La Salle Bldg., OALR
SMITH, ARTHUR E.....Donaldson Bldg., OALR
STROUT, E. S.....74 S. 11th St., OALR
STROUT, G. E.....Donaldson Bldg., OALR
WATSON, JAMES A.....74 S. 11th St.,
WOOD, DOUGLAS.....Donaldson Bldg., OALR
WRIGHT, C. DARCY.....307 Yeats Bldg., OALR

NEW ULM

REINEKE, GEO. F.....OALR

ROCHESTER

ANDERSON, CARL M.....Mayo Clinic, ALR
BENEDICT, WILLIAM L.....Mayo Clinic, Op.
FIGI, F. A.....Mayo Clinic, LR
HEMPSTEAD, BERT E.....Mayo Clinic, ALR
LILLIE, HAROLD I.....Mayo Clinic, ALR
NEW, GORDON B.....Mayo Clinic, L
PRANGEN, AVERY DE H.....Mayo Clinic, Op.
STARK, WM. B.....Mayo Clinic, ALR
WAGENER, HENRY P.....Mayo Clinic, Op.

ST. PAUL

BERRISFORD, PAUL D.....Lowry Bldg., OALR
BINGER, H. E.....823 Lowry Bldg., OALR
BOECKMANN, EGIL.....Lowry Bldg., OALR
BRAY, E. R.....342 Lowry Bldg., OALR
BROWN, JOHN C.....632 Lowry Bldg., OALR
BURCH, FRANK E.....754 Linwood Pl., Op
CONNOR, CHAS. E.....Hamm Bldg., ALR
FOGARTY, CHAS. W.....1118 Lowry Bldg., OALR
FULTON, JOHN F.....833 Lowry Bldg., OALR
LARSEN, CARL L.....1027 Lowry Bldg., OALR
LEWIS, WILLIAM W.....836 Lowry Bldg., OALR
MALONEY, THOMAS J.....1032 Lowry Bldg., OALR
NELSON, LOUIS A.....830 Lowry Bldg., Op.
ROTHSCHILD, HAROLD J.....239 Lowry Bldg., OALR
SHELLMAN, J. L.....Lowry Bldg., OALR
WHEELER, MERRITT W.....1027 Lowry Bldg., OALR
WOLD, K. C.....Med. Arts Bldg., OALR

WINONA

RISSER, EDWARD D.....310 Choate Bldg., OALR
WALKER, GEO. H.....172 Main St., OALR

MISSISSIPPI

GREENVILLE

MONTGOMERY, DANIEL C..... OALR

GULFPORT

MCWILLIAMS, CHAS. A..... ALR

NATCHEZ

GAUDET, LUCIAN S.....405 Franklin St., OALR

OXFORD

GUYTON, B. S..... OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., Op.
EDWARDS, C. J.....First National Bank Bldg., OALR
HOWARD, E. F.....First National Bank Bldg., ALR
JONES, EDLEY H.....First National Bank Bldg., OALR

MISSOURI

CARTHAGE

POST, WINFRED B.....First National Bank Bldg., OALR
POWER, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

SNEED, CARL M.....506 W. Broadway, OALR

KANSAS CITY

ALLEN, CHAS. E.....712 Waldheim Bldg., OALR
ALTRINGER, A. N.....710 Rialto Bldg., ALR
BEAL, HOMER A.....1002 Argyle Bldg., ALR
BEIL, J. W.....Argyle Bldg., OALR
BELLOWS, GEORGE E.....Rialto Bldg., Op.
BLAKESLEY, T. S.....Lathrop Bldg., OALR
CURDY, R. J.....1134 Rialto Bldg., Op.
CURRAN, E. J.....1805 Fed. Res. Bk. Bldg., Op.
DAVIS, HARRY B.....712 Bryant Bldg., OALR
DIXON, OTTO J.....902 Med. Arts Bldg., ALR
EUBANK, AMBROSE E.....636 Argyle Bldg., OALR
FOSTER, HAL.....1014 Chambers Bldg., ALR
HOWARD, J. W.....Rialto Bldg., OALR
KEITH, W. E.....Waldheim Bldg., OALR
LANING, J. H.....907 Rialto Bldg., ALR
LEA, JOS. A.....1125 Rialto Bldg., ALR
LEMOINE, A. N.....1118 Rialto Bldg., Op.
LICHTENBERG, J. S.....Rialto Bldg., Op.
LORIE, ALVIN.....Commerce Bldg., ALR
LUX, PAUL.....409 Commerce Bldg., ALR
MCALESTER, A. W., JR.....625 Bryant Bldg., Op.
MCCARTY, VIRGIL W.....800 Rialto Bldg.

McKee, Joseph W.....1222 Rialto Bldg., Op.
Miller, Hugh.....Chambers Bldg., OALR
Moss, Harvey E.....1321 Rialto Bldg., OALR
Mott, John S.....214 Argyle Bldg., OALR
Myers, John L.....815 Shukert Bldg., OALR
Painter, A. M.....418 Bryant Bldg., ALR
Roberts, S. E.....1810 Federal Reserve Bank Bldg., ALR
Russell, E. L.....700 Argyle Bldg., OALR
Schutz, W. H.....Bryant Bldg., OALR
Shoemaker, Ferdinand,
U. S. Vet. Hosp., No. 67, 27th and Paseo Sts., OALR
Shumate, D. L.....500 Argyle Bldg., OALR
Thomason, H. E.....Rialto Bldg., ALR
Teall, Raymond E.....1100 Rialto Bldg., ALR
Yazel, Herman E.....733 Rialto Bldg., OALR

ST. JOSEPH

Forgrave, L. Robert.....204 Logan Bldg., OALR
Kenney, Wm. L.....Corby Forsee Bldg., OALR
Minton, Wm. H.....1st State Bk., Bldg., OALR
Proud, Willard C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

Alden, Arthur M.....537 Frisco Bldg., ALR
Alvis, Bennett Y.....900 Carleton Bldg., Op.
Arbuckle, Millard F.....1011 Beaumont Med. Bldg., ALR
Ball, James Moores.....4500 Olive St., Op.
Barck, Carl.....Humboldt Bldg., Op.A
Bardenheier, F. G. A.....Frisco Bldg., Op.A
Black, W. D.....Metropolitan Bldg., ALR
Brown, Clyde O.....537 N. Grand Blvd., ALR
Burns, S. S.....Missouri Theatre Bldg., ALR
Charles, Jos. W.....3720 Washington Ave., Op.
Costen, J. B.....3720 Washington Ave., ALR
Davis, Charles L.....3542 Washington Ave., ALR
Dean, L. W.....Washington Univ., ALR
Donnell, N. R.....Metropolitan Bldg., Op.
Dyer, C. P.....408 Humboldt Bldg., Op.
Eber, Carl T.....1006 Carleton Bldg., Op.
Eimer, Charles E.....316 Lister Bldg., ALR
Ewing, Arthur E.....Metropolitan Bldg., Op.
Ferris, David P.....1000 Carleton Bldg., ALR
Freimuth, L. E.....234 University Club Bldg., ALR
Glick, Harry N.....3115 S. Grand Ave., ALR
Goldstein, M. A.....3858 Westminster Pl., ALR
Green, John.....3720 Washington Blvd., Op.
Guggenheim, L. K.....Carleton Bldg., ALR
Gundelach, C. A.....825 University Club Bldg., ALR
Hansel, French Keller.....Beaumont Bldg., ALR
Hardesty, J. F.....1021 Missouri Bldg., Op.
Hardy, W. F.....Humboldt Bldg., Op.

HOURN, GEORGE E.....511 University Club Bldg., ALR
HOWARD, FRANCIS C.....607 N. Grand Blvd., ALR
HOWARD, HARVEY.....School of Med., Washington Univ., Op.
JACOBS, MAX W.....Missouri Theatre Bldg., Op.
JENNINGS, J. ELLIS.....807 Carleton Bldg., Op.
JONES, VINCENT L.....3511 Washington Ave., Op.
KELLEY, ISAAC D., JR.....811 Beaumont Med. Bldg., ALR
LAMB, HARVEY.....826 Metropolitan Bldg., Op.
LEAVY, C. A.....Metropolitan Bldg., ALR
LUEDDE, W. H.....Metropolitan Bldg., Op.
LYMAN, HARRY W.....700 Carleton Bldg., ALR
MCMAHON, BERNARD JOHN.....Missouri Theatre Bldg., ALR
MASON, ROY E.....422 Frisco Bldg., Op.
MAYER, LEO L.....900 Carleton Bldg., Op.
MILLER, VERNON B.....810 Metropolitan Bldg., ALR
MYERS, EUGENE LEE.....Wall Bldg., ALR
PAYNE, R. J.....University Club Bldg., ALR
PFINGSTEN, C. F.....820 Metropolitan Bldg., ALR
POOS, GROVER H.....301 Univ. Club Bldg. OALR
POST, LAWRENCE T.....520 Metropolitan Bldg., Op.
POST, M. HAYWARD.....520 Metropolitan Bldg., Op.
PROETZ, A. W.....Beaumont Bldg., ALR
REIM, HUGO.....417 Metropolitan Bldg., Op.
RYAN, L. M.....Wall Bldg., ALR
SAUER, W. E.....Humboldt Bldg., ALR
SCHOLZ, R. P.....Metropolitan Bldg., ALR
SCHWARTZ, FREDERICK O.....701 Metropolitan Bldg., Op.
SENSENEY, EUGENE T.....316 Lister Bldg., ALR
SHAHAN, W. E.....520 Metropolitan Bldg., Op.
SHOEMAKER, W. A.....1006 Carleton Bldg., Op.
SMIT, WM. M.....719 Missouri Theatre Bldg., ALR
TOOKER, C. W.....Carleton Bldg., Op.
VAN METER, EUGENE R.....4473 Washington Ave., ALR
WESTLAKE, SAMUEL B.....Beaumont Med. Bldg., ALR
WIENER, MEYER.....900 Carleton Bldg., Op.
WOOD, V. VISSCHER.....201 Humboldt Bldg., ALR
WOODRUFF, FREDERICK E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, JOHN G.....Ilgenfritz Bldg., OALR
TITSWORTH, GUY.....111 W. 4th St., OALR

SPRINGFIELD

BAILEY, HAROLD.....840 Landers Bldg., OALR
COFFELT, THEODORE A.....508 Woodruff Bldg., OALR
KLINGER, THOS. O.....318 Landers Bldg., OALR
SMITH, CARLIE SOUTER.....214 N. Jefferson Ave., OALR

TRENTON

AMBROSE, ELMER C.....910½ Main St., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....Electric Light Bldg., OALR

BUTTE

DONOVAN, JNO. A.....507 Phoenix Bldg., OALR
MORSE, A. W.....507 Phoenix Bldg., OALR
POTTER, PETER.....125 W. Granite St., OALR

GREAT FALLS

COULTER, CHAS. F.....First Natl. Bank Bldg., OALR

LEWISTOWN

PARSONS, J. G.....313 Bank Electric Bldg., OALR

ROUNDUP

LEWIS, GEORGE A.....OALR

NEBRASKA

BEATRICE

TUCKER, JAMES C.....114 S. 6th St., OALR

COLUMBUS

CAMPBELL, C. H.....Col. State Bk. Bldg., OALR

KEARNEY

FOX, CHAS. H.....2119 Central Ave., OALR

LINCOLN

BLACK, PAUL.....514 1st Natl. Bk. Bldg., Op.
BROOKS, EARL B.....707 1st Natl. Bk. Bldg., OALR
CURTIS, W. L.....1503 S. 22nd St., OALR
DAYTON, W. L.....302 Funke Bldg., OALR
FURGASON, A. P.....Richards Block, OALR
HOMPES, J. J.....Security Mutual Bldg., OALR
SANDERSON, DAVID D.....Funke Bldg., OALR
THOMAS, JAMES W.....726 Security Mutual Bldg., OALR
ZEMER, S. G.....726 Security Mutual Bldg., OALR

NORFOLK

GADBOIS, ARTHUR E.....421 Norfolk Ave., OALR
NELSON, LLOYD I.....Ommerman Bldg., OALR

OMAHA

BANISTER, COL. J. M.....920 Med. Arts Bldg., OALR
BUSHMAN, L. B.....631 City Natl. Bk. Bldg., OALR
CALLFAS, W. F.....1620 Med. Arts Bldg., ALR
DAVIS, JOHN C., JR.....425 Aquila Court, OALR
FAIRCHILD, NORA M.....Med. Arts Bldg., Op.
GIFFORD, HAROLD.....Med. Arts Bldg., Op.
GIFFORD, S. R.....Med. Arts Bldg., Op.
HANEY, WM. P.....1500 Med. Arts Bldg., OALR

KNODE, A. R.....708 Med Arts Bldg., ALR
KULLY, B. M.....Med. Arts Bldg., ALR
LEMERE, H. B.....Med. Arts Bldg., OALR
OWEN, F. S.....Med. Arts Bldg., OALR
PATTON, J. M.....Med. Arts Bldg., Op.
POTTER, GEO. B.....Med. Arts Bldg., OALR
POTTS, J. B.....Med. Arts Bldg., ALR
RUBENDALL, C.....Med. Arts Bldg., OALR
SWAB, CHARLES M.....Med. Arts Bldg., Op.
UREN, C. T.....537 City National Bank Bldg., ALR
WHERRY, W. P.....1500 Med. Arts Bldg., OALR

NEVADA

RENO

FULLER, JOHN A..... OALR

NEW HAMPSHIRE

NASHUA

NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

FISHER, JAMES A.....Fitkin Bldg., OALR
UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. C.....124 S. Illinois Ave., ALR
MCGIVERN, C. S.....101 S. Indiana Ave., ALR

BLOOMFIELD

MOORE, JOHN D.....6 Washington St., OALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

JERSEY CITY

PYLE, WALLACE.....15 Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

BEGGS, W. F.....671 Broad St., OALR
CHATTIN, J. FRANKLIN.....671 Broad St., OALR
HUGHES, L. W.....1019 Broad St., OALR
HURFF, J. W.....86 Washington St., OALR
KEIM, WILLIAM F.....25 Roseville Ave., ALR
O'CONNOR, D. F.....671 Broad St., OALR
ORTON, HENRY B.....24 Commerce St., ALR
QUINBY, W. O'G.....14 James St., OALR

SHERMAN, ELBERT S.....671 Broad St., Op.
WOOD, EARL LER.....33 Lincoln Park, ALR
ZEHNDER, A. CHARLES.....188 Roseville Ave., OP.

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

TRENTON

ADAMS, C. F.....34 W. State St., OALR
CRANE, J. WELLINGTON.....128 Third Ave.,OALR

UNION CITY

SACCO, A. G.....440 New York Ave., OALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON I.....1st Natl. Bk. Bldg., OALR

NEW YORK

ALBANY

BEDELL, ARTHUR J.....344 State St., Op.
DOWLING, J. IVIMEY.....116 Washington Ave., OALR
HINMAN, E. E.....146 Washington Ave., ALR
JUDGE, HARRY V.....46 Willett St., Op.

BINGHAMTON

KINNE, SANFORD H.....73 Main St., ALR
RANDALL, HIRAM.....81 Main St., ALR
WATSON, H. D.....151 Front St., OALR

BROOKLYN

ARROWSMITH, HUBERT.....11 Schermerhorn St., ALR
EVANS, J. N.....23 Schermerhorn St., Op.
GOLDING, JOSEPH E.....170 Hancock St., Op.
GREENFIELD, SAMUEL D.....169 New York Ave., ALR
HANCOCK, J. C.....135 Cambridge Pl., Op.
HARGITT, CHARLES A.....17 Schermerhorn St., Op.
KRIMSKY, JOSEPH.....263 Eastern Parkway, OALR
KRUSKAL, I. D.....863 Prospect Pl., Op.
LLOYD, RALPH I.....14 Eighth Ave., Op.
LOMBARDO, MELCHIORE.....Med. Arts Bldg., Op.
MYERSON, M. C.....30 E. 4th St., ALR
ROGERS, ROBERT M.....680 St. Johns Pl., Op.

BUFFALO

ANDREWS, H. D.....33 Allen St., Op.
BENNETT, ARTHUR G.....150 N. Pearl St., Op.
BEYER, LOUIS J.....449 Delaware Ave., ALR
BLAAUW, E. E.....190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE.....468 Delaware Ave., ALR
BROWN, C. M.....510 Delaware Ave., ALR

COTT, C. C.....483 Delaware Ave., ALR
COWPER, H W.....543 Franklin St., Op.
EDSON, R. A.....498 Delaware Ave., Op.
FAIRBAIRN, JOHN F.....503 Delaware Ave., ALR
FLAGG, J. D.....473 Virginia St., Op.A
GLOSSER, HERBERT H.....448 Franklin St., OALR
HEALY, JOHN F.....503 Delaware Ave., ALR
KOENIG, IVAN J.....40 North St., Op.
LEWIS, F. PARK.....454 Franklin St., Op.
MCKEE, OTTO S.....131 Linwood Ave., ALR
MARCH, CLARA A.....465 Ashland Ave., Op.
PHILLIP, WM. LINTON.....131 Linwood Ave., Op.
RENNER, W. S.....341 Linwood Ave., ALR
WEED, HARRY M.....196 Linwood Ave., Op.
WURTZ, W. J. M.....528 Delaware Ave., ALR

CORTLAND

HIGGINS, R. P.....20 Court St., OALR

DUNKIRK

JACKLE, ARTHUR F.....63 E. 4th St., Op.

GENEVA

SPENGLER, JOHN ARTHUR.....423 S. Main St., Op.

GLENS FALL

FIELDING, F. G.....70 Ridge St., ALR

JAMESTOWN

REGER, H. S.....15 E. Fourth St., OALR

KEW GARDENS

FERNLUND, GEORGE B.....Kew Arms, OALR

LOCKPORT

RINGUEBERG, E. N. S.....13 Main St., Op.

MT. VERNON

THOMSON, J. J.....Esplanade Gardens, E. LincolnAve., ALR

NEW ROCHELLE

BECK, A. L.....421 Huguenot St., OALR
WOODRUFF, WALTER STUART.....421 Huguenot St., ALR

NEW YORK CITY

ALMOUR, R. W.....51 W. 73rd St., ALR
AUERBACH, JULIUS.....120 W. 86th St., ALR
BALLIN, M. J.....20 E. 76th St., ALR
BEACH, B. S.....76 W. 86th St., Op.
BERENS, CONRAD.....30 E. 40th St., Op.
BRUDER, J.....229 W. 78th St., OALR
CARTER, W. W.....2 E. 54th St., ALR

COHEN, MARTIN.....1 W. 85th St., Op.
COHN, FELIX.....47 W. 87th St., ALR
COTT, D. N.....141 St. Marks Pl., St. George, Staten Isl., ALR
CUNNING, DANIEL S.....114 E. 54th St., OALR
DAVIS, A. EDWARD.....40 E. 61st St., Op.
DAVIS, G. E.....17 W. 74thSt., OALR
DIXON, G. S.....218 Second Ave., R
DOHERTY, W. B.....150 W. 55th St., Op.
DUNNINGTON, J. H.....30 W. 59th St., Op.
ELLIS, Z. H.....30 W. 59th St., Op.
FRANKEL, EDWARD, JR.....152 W. 58th St., ALR
FREUDENTHAL, WOLFF.....24 W. 88th St., ALR
GATEWOOD, W. L.....330 Park Ave., ALR
GLOGAU, O.....1130 Park Ave., Cor. 91st St., ALR
GOLDMAN, A MILTON.....121 E. 60th St., Op.
GOLDMAN, H. G.....57 W. 58th St., ALR
GOTTLIEB, MARK J.....23 W. 73rd St., ALR
GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
GUTTMAN, J.....987 Madison Ave., OALR
HALLETT, G. DEWAYNE.....274 W. 86th St., Op.
HARDY, LEGRAND H.....30 E. 40th St., Op.
HARTSHORNE, ISAAC.....30 W. 59th St., Op.
HASKINS, W. H.....57 W. 57th St., ALR
HAYS, HAROLD M.....22 W. 74th St., ALR
HETRICK, L. E.....40 E. 61st St., ALR
HOPKINS, W. E.....515 Park Ave., OALR
HURD, L. M.....39 E. 50th St., ALR
HUTCHINSON, ALBERT T.....30 W. 59th St., ALR
IMPERATORI, C. J.....18 E. 38th St., ALR
INGERMAN, S. M.....418 Central Park, W., OALR
IRWIN, FRANK N.....114 E. 54th St., Op.
JACOBS, S. M.....1018 E. 163rd St., OALR
JARECKY, HERMAN.....168 W. 86th St., OALR
JOHNSON, THOMAS HOLLAND.....30 W. 59th St., Op.
JUDD, H. B.....47 E. 57th St., ALR
KELLY, JOSEPH D.....114 E. 54th St., OALR
KEY, BEN WITT.....100 W. 59th St., Op.
KING, J. J.....30 E. 40th St., ALR
KIRBY, D. B.....30 W. 59th St., Op.
KNAPP, ARNOLD.....10 E. 54th St., Op.
KOPETZKY, S. J.....51 W. 73rd St., ALR
KRUG, ERNST F.....12 W. 44th St., Op.
LA VIGNE, A. A.....471 Park Ave., OALR
LUBMAN, MAX.....17 E. 96th St., ALR
MCCOY, JOHN.....730 Fifth Ave., ALR
MCDANNALD, C. E.....100 W. 59th St., Op.
MCDONALD, DENNIS J.....17 E. 38th St., ALR
MAY, CHAS. H.....698 Madison Ave., Op.
MAYBAUM, J. L.....1079 Park Ave., ALR
MAYER, EMIL.....666 West End Ave., ALR
MEIERHOF, E. L.....12 E. 86th St., OALR

MINSKY, HENRY.....215 W. 88th St., Op.
MITTENDORF, A. D.....399 Park Ave., Op.
MITTENDORF, W. KEITH.....115 E. 53rd St., Op.
MORRISON, WM. WALLACE.....39 E. 50th St., ALR
NISSELSON, MAX.....754 Beck St., ALR
NORTHCOTT, THOMAS A.....17 E. 38th St., Op.
PALMER, ARTHUR.....667 Madison Ave., ALR
PETERSON, R. A.....150 Fifth Ave., OALR
PHILLIPS, W. C.....30 E. 58th St., ALR
REESE, ALGERNON B.....50 W. 52nd St., Op.
SCAL, JOSEPH COLEMAN.....44 W. 74th St., ALR
SCHILLER, A. N.....249 West End Ave., ALR
SCHLIVEK, KAUFMAN.....1016 Fifth Ave., Op.
SCHWARTZ, A. A.....203 W. 90th St., ALR
SOMBERG, J. S.....60 E. 67th St., Op.
STARK, JESSE B.....45 Park Ave., Op.
STURGES, LEIGH F.....121 E. 60th St., ALR
WEEKS, WEBB WILLIAM.....20 E. 53rd St., Op.
WHEELER, J. M.....30 W. 59th St., Op.
WIENER, ALFRED.....550 Park Ave., OALR
YANKAUER, SIDNEY.....121 E. 60th St., ALR

NIAGARA FALLS

PRICE, NORMAN W.....445 Third St., OALR
ROOKER, A. M.....225 Gluck Bldg., ALR

NYACK

TOMS, S. W. S.....120 S. Broadway, OALR

OLEAN

TINDOLPH, L. W.....First Natl. Bank Bldg., ALR

ONEIDA

CROCKETT, R. L.....Madison Co. Trust Bldg., Op.

PLATTSBURG

SCHNEIDER, ANTON S.....56 Court St., Op.A

POUGHKEEPSIE

KRIEGER, WM. A.....35 Market St., OALR

ROCHESTER

BARBER, F.....75 S. Fitzhugh St., Op.
CARROLL, GEO. G.....614 W. Main St., OALR
CLARK, L. H.....337 Monroe Ave., OALR
GIPNER, J. F.....53 Fitzhugh St. Op.
JONES, L. W.....53 S. Fitzhugh St., OALR
KENNEDY, ELDRED W.....Gas & Electric Bldg., Op.
LERNER, MACY LEVI.....332 Park Ave., Op.
MCDOWELL, N. D.....275 Alexander St., OALR
MORRIS, A. G.....303 Alexander St., ALR

SHAPERO, I. M.....289 Alexander St., OALR
SNELL, ALBERT C.....53 S. Fitzhugh St., Op.

SYRACUSE

BRITTEN, G. S.....University Blk., ALR
BROWN, MORTIMER G.....802 University Bldg., ALR
BRUST, H. O.....820 S. Crouse Ave., ALR
FOWLER, S. R.....University Bldg., OALR
GILLETTE, D. F.....300 University Blk., Op.
HALL, EDWIN P.....State Tower Bldg., ALR
JOY, HAROLD HENRY.....504 State Tower Bldg., Op.
KAUFFMAN, A. B.....Gurney Bldg., ALR
KLINE, H. G.....State Tower Bldg., ALR
MARLOW, SEARLE B.....1004 State Tower Bldg., Op.
MOORE, ROY S.....405 E Fayette St., ALR

TROY

SULZMAN, FRANK MALCOLM.....1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H.....258 Genesee St., OALR

WATERTOWN

ATKINSON, WALTER S.....168 Sterling St., Op.

WHITE PLAINS

MORIARTY, RICHARD W.....38 S. Broadway, OALR

WOODHAVEN

FROTHINGHAM, EDWARD.....8644 94th St., OALR

NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H.....101 Haywood St., OALR
GREENE, JOSEPH BERRY.....305 Haywood Bldg., ALR
MCCLELLAN, W. E.....101 Haywood St., ALR

CHARLOTTE

MATHESON, J. P.....6 W. 7th St., ALR
PEELER, C. N.....W. 7th St., OALR
SLOAN, HENRY L.....6 W. 7th St., Op.
WHISNANT, A. M.....517 Professional Bldg., OALR

GREENSBORO

REAVES, CHAS. R...Reaves Infirmary, 117 W. Sycamore St., OALR
REAVES, W. P.....Reaves Infirmary, 117 W. Sycamore St., OALR

MOREHEAD CITY

JOHNSON, OTIS H.....OALR

NEW BERN

DANIELS, R. L.....Elks Bldg., OALR

RALEIGH

HICKS, VONNIE MONROE.....Professional Bldg., Op.
WRIGHT, J. B.....Professional Bldg., ALR

SALISBURY

BRAWLEY, R. V.....Wallace Bldg., Op.

WILMINGTON

MURPHY, J. G.....Murchison Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W.....210 O'Hanlon Bldg., OALR

NORTH DAKOTA

FARGO

RINDLAUB, ELIZABETH P.....620 Front St., OALR
RINDLAUB, J. H.....620 Front St., OALR
RINDLAUB, M. P., JR.....620 Front St., OALR

MINOT

MCCANNELL, A. D.....20 Central Ave., OALR

WAHPETON

RICE, CLARENCE PRENTICE..... OALR

OHIO

AKRON

BROWN, L. E.....2nd Natl. Bank Bldg., OALR
MOORE, T. K.....416 Central Sav. & Trust Bldg., OALR
STEVENSON, D. W.....165 E. Market St., OALR

ALLIANCE

KING, G. L.....537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H..... OALR
HARBERT, JOHN P.....135 N. Main St., OALR

CANTON

HOFFMAN, JOHN N.....308 Brant Bldg., ALR
SCHILD, E. H.....Brant Bldg., Op.

CINCINNATI

ABRAHAMSON, IRA.....605 Traction Bldg., Op.
ASBURY, MARY K.....707 Race St., Op.
BEYER, A. G.....19 Garfield Pl., OALR
BROWN, ALBERT LOUIS.....2600 Union Central Bldg., Op.
GOODYEAR, H. M.....556 Doctors Bldg., ALR
HANS, C. L.....19 W. 7th St., OALR
HARRIS, W. C.....7th & Race Sts., ALR

IGLAUER, SAMUEL.....707 Race St., ALR
JONES, C. C.....656 Doctors Bldg., ALR
KING, CLARENCE.....Union Central Bldg., Op.
KING, ED.....707 Race St., Op.
LYLE, DONALD J.....19 W. 7th St., OALR
MITHOEFER, W.....19 Garfield Pl., ALR
MURPHY, W. E.....7th and Race Sts., OALR
PHINNEY, F. D.....22 W. 7th St., OALR
RAY, VICTOR.....19 W. 7th St., Op.
SIEGEL, FRANCIS X.....904 Union Central Life Bldg., OALR
STANBERRY, HENRY.....556 Doctors Bldg., Op.
STEWART, T. M.....901 Union Trust Bldg., OALR
STITT, H. L.....656 Doctors Bldg., ALR
TANGEMAN, H. F.....606 Union Central Life Bldg., ALR
THOMPSON, E. H.....8 W. 9th St., OALR
URNER, M. H.....2600 Union Central Bldg., OALR
VAIL, DERRICK T.....24 E. 8th St., Op.
VAIL, H. H.....24 E. 8th St., ALR

CLEVELAND

ABBOTT, W. J.....8314 Euclid Ave., OALR
BRUNER, ABRAM BRENEMAN.....1214 Guardian Bldg., OALR
BRUNER, WM. E.....Guardian Bldg., Op.
BURKE, T. A.....Rose Bldg., OALR
COGAN, J. E.....707 Rose Bldg., OALR
CUTLER, FRANKLIN E.....1026 Schofield Bldg., ALR
FORSYTHE, S. T.....235 Colonial Arcade, OALR
KENDALL, M. R.....Osborn Bldg., ALR
LARGE, S. H.....Hanna Bldg., ALR
LENKER, JOHN N.....1020 Huron Rd., ALR
MCDONALD, C. L.....964 Rose Bldg., OALR
METZ, ROY B.....1214 Guardian Bldg., Op.
METZENBAUM, MYRON.....Rose Bldg., ALR
MONSON, S. H.....1322 B. F. Keith Bldg., OALR
MULLIN, W. V..Cleveland Clinic, Euclid Ave., and 93rd St., ALR
MUSSUN, W. G.....610 Hanna Bldg., ALR
NELSON, CHAS. F.....606 Schofield Bldg., OALR
PITKIN, C. E.....Osborn Bldg., ALR
PRENDERGAST, D. A.....850 Hanna Bldg., OALR
QUITTNER, S. S.....3912 Prospect Ave., OALR
RIEGER, W. H.....Hanna Bldg., ALR
RUST, E. G.....Hanna Bldg., Op.A
SHACKLETON, W. E.....2323 Prospect Ave., Op.
SHIRAS, HOWARD H.....629 Guardian Bldg., Op.
SHIREY, O. M.....Osborn Bldg., ALR
SIMONDS, O. F.....Hanna Bldg., Op.
STOTTER, ARTHUR L.....1148 Euclid Ave., OALR
TRIPP, IRA A.....436 Rose Bldg., OALR
TUCKERMAN, W. C.....733 Osborn Bldg., Op.
TUCKERMAN, W. H.....733 Osborn Bldg., OALR
WAUGH, I. M.....Cleveland Clinic, Euclid Ave. at 93rd St., OALR

COLUMBUS

BEATTY, H. G.....681 E. Broad St., ALR
BROWN, JOHN E.....370 E. Town St., OALR
CLARK, I. G.....188 E. State St., OALR
FROST, ALBERT D.....681 E. Broad St., Op.
HAUER, A. M.....327 E. State St., ALR
PROUT, A. W.....370 E. Town St., OALR
SANOR, DANIEL G.....206 E. State St., ALR
SILBERNAGEL, C. E.....283 E. State St., OALR
STOUTENBOROUGH, WM. A.....313 E. State St., OALR
SULZER, G. A.....327 E. State St., OALR
THOMAS, FRANCIS W.....327 E. State St., OALR
TIMBERMAN, ANDREW.....318 E. State St., OALR
WRIGHT, J. W.....Central National Bldg., Op.

DAYTON

BINKLEY, ROY S.....530 Fidelity Bldg., OALR
BLACKBURN, W. J.....663 Reibold Bldg., OALR
DUTROW, H. V.....1040 Fidelity Bldg., OALR
FARMER, A. G.....Fidelity Bldg., OALR
FOUTS, JOHN D.....910 Fidelity Bldg., OALR
HARRIS, H. B.....Fidelity Medical Bldg., OALR
KILBOURNE, P. H.....Fidelity Medical Bldg., OALR
MILLETTE, J. W.....Reibold Bldg., OALR

ELYRIA

GILL, GEORGE.....Elyria Sav. & Trust Bldg., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts., OALR

LIMA

STUEBER, F. G.....209 W. North St., OALR
STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....O. P. S. Bldg., OALR

MANSFIELD

GARBER, J. M.....14 N. Mulberry St., OALR
WISE, RALPH C.....OALR

MARIETTA

SAUER, W. W.....Box 416, OALR

MARION

BRICKLEY, DANIEL W.....196 W. Center St., OALR

MASSILLON

SCOTT, MERLE E.....1st Natl. Bk. Bldg., OALR

MIDDLETOWN

WILLIAMS, WALTON H.....Castell Bldg., OALR

NEWARK

HATCH, C. B.....3 W. Church St., OALR

SPRINGFIELD

EASTON, J. C.....1st Natl. Bk. Bldg., OALR
HARTLEY, F. A.....302 E. High St., OALR
HOGUE, D. W.....1007 Tecumseh Bldg., OALR
MINOR, C. L.....1st Natl. Bk. Bldg., OALR

STEUBENVILLE

GOURLY, G. F.....Sinclair Bldg., OALR

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDERDYCE, WM.....513 Madison Ave., OALR
ALTER, F. W.....316 Michigan St., OALR
DENMAN, IRA O.....425 Ohio Bldg., OALR
KING, CHAS. R.....471 Spitzer Bldg., OALR
LUKENS, CHARLES.....316 Michigan St., OALR
SNYDER, W. H.....220 Michigan St., OALR
STEINFELD, A. L.....125–15th St., OALR
WINGER, IRA B.....225 Michigan St., OALR

WARREN

STIMSON, GEORGE W.....Packard Apt., OALR

XENIA

MADDEN, REED.....Allen Bldg., OALR
SHIELDS, LAWRENCE.....Allen Bldg., ALR

YOUNGSTOWN

BEARD, H. J.....510 Dollar Bank Bldg., OALR
GIBSON, R. D.....310 Wick Ave., OALR
HARTZEL, S. M.....Dollar Bank Bldg., OALR

ZANESVILLE

CULBERTSON, L. R.....Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D.....Cent. Natl. Bk. Bldg., OALR

ENID

PIPER, ARTHUR S.....Enid Clinic., OALR

GUTHRIE

BARKER, CHAS. B.....224½ W. Okla. Ave., OALR

McALESTER

DAVIS, J. E.....Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M.....404 Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E.....503 Medical Arts Bldg., OALR
FERGUSON, E. S.....902 Medical Arts Bldg., OALR
GUTHRIE, A. L.....1010 Medical Arts Bldg., OALR
MCHENRY, D. D.....604 Medical Arts Bldg., OALR
NEWTON, L. A.....609 Medical Arts Bldg., OALR

TULSA

BRASWELL, JAMES C.....Med. Arts Bldg., OALR
COOK, W. ALBERT.....506 Palace Bldg., OALR
ROTH, ALBERT W.....Med. Arts Bldg., OALR

OREGON

LA GRANDE

BOUVY, L. B.....Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE.....Sellings Bldg., OALR
BOUVY, HARRY.....800 Pittock Blk., OALR
BRUERE, GUSTAVE E.....Journal Bldg., OALR
CARRUTH, HOWARD E.....716 Med. Arts Bldg., OALR
DAVIS, RALPH F.....Mayer Bldg., OALR
DICKSON, JOHN F.....418 Mayer Bldg., OALR
FENTON, RALPH A.....806 Medical Arts Bldg., OALR
HENTON, G. E.....Morgan Bldg., OALR
JOHNSTON, WILSON.....611 Lovejoy St., OALR
KARKEET, ROBERT B.....1203 Selling Bldg., OALR
KIEHLE, F. A.....410 Medical Arts Bldg., OALR
KISTNER, F. B.....471 Montgomery Drive, OALR
LUCAS, HAROLD R.....411 Selling Bldg., OALR
LUPTON, IRVING M.....Medical Arts Bldg., OALR
MCCOOL, J. L.....909 Stevens Bldg., Op.
WEEKS, JOHN E.....Dunthorpe, Rt. 8., Op.

PENNSYLVANIA

ALLENTOWN

HARDING, FREDERICK B.....Med. Arts Bldg., OALR

ALTOONA

GLOVER, S. P.....1118–12th Ave., OALR

BEAVER

CRUMRINE, NORMAN R.....595 3rd St., OALR
SCROGGS, J. J.....218 E. 3rd St., Op.

BETHLEHEM

CHASE, W. D.....230 E. Broad St., ALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....357 N. Main St., OALR
HAZLETT, L. R.....Savings & Trust Bldg., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR

CHESTER

CROSS, G. H.....525 Welsh St., Op.

CONNELLSVILLE

HARRISON, FRED H.....308 1st Natl. Bk. Bldg., OALR

ERIE

DENNIS, DAVID N.....221 W. 9th St., Op.
DENNIS, EDWARD P.....309 W. 10th St., OALR
FUST, J. H. E.....154 W. 8th St., OALR
HEARD, C. F.....813 Sassafras St., OALR
JACKSON, J. D.....232 W. 8th St., OALR
RUSSELL, J. A. MERLE.....813 Sassafras St., ALR
SCHLINDWEIN, G. WM.....138 W. 9th St., OALR
SHREVE, OWEN M.....162 W. 8th St., Op.

FRANKLIN

JOBSON, G. B.....Printz Bldg., OALR

GREENSBURG

FAIRING, JOHN W.....206 Tribune Review Bldg., OALR
McKEE, CLAUD W.....Coulter Bldg., OALR

HARRISBURG

FARNSLER, H. HERSHEY.....1438 Market St., OALR
GRACEY, GEO. F.....209 State St., OALR
PARK, J. WALTER.....32 N. 2d St., OALR
REBUCK, C. S.....412 N. 3d St., OALR

HAZELTON

REICHE, O. C.....Markle Bk. Bldg., OALR

HUNTINGDON

SEARS, W. H.....514 Penn St., OALR

JOHNSTOWN

BARKER, O. G. A.....804 Johnstown Trust Bldg., Op.
HARRIS, C. M.....604 Johnstown Trust Bldg., OALR
RUSH, C. C.....907 U. S. Natl. Bk. Bldg., OALR

LEBANON

REESE, MONROE D.....123 N. 8th St., OALR

MIDDLETOWN

GEORGE, HENRY W.....19 N. Union St., Op.

NEW KENSINGTON

McConnell, T. E.....Logan Trust Bldg., OALR
Steim, J. M.....1st Natl. Bk. Bldg., Op.

NORTH EAST

Heard, Mary K.....41 W. Main St., Op.

PHILADELPHIA

Alexander, Geo. J.....1629 Spruce St., ALR
Annon, W. T.....235 S. 15th St., ALR
Appleman, L. F.....308 S. 16th St., Op.
Babbitt, James A.....1912 Spruce St., ALR
Baldwin, Kate W.....1117 Spruce St., ALR
Balentine, P. L.....1524 Chestnut St., Op.
Blair, Mortimer W.....N. E. Cor. 20th & Chestnut Sts., Op.
Boehringer, W. H.....700 Central Medical Bldg., OALR
Brown, Samuel Horton.....1901 Mt. Vernon St., Op.
Brumm, Seth A.....Med. Arts Bldg., OALR
Butler, Margaret F.....1831 Chestnut St., ALR
Butt, Miriam M.....2045 Chestnut St., Op.
Clerf, Louis Henry.....128 S. 10th St., ALR
Coates, Geo. M.....1721 Pine St., ALR
Cohen, Herman B.....Spruce and 13th Sts., ALR
Cohen, Samuel.....2009 Pine St., ALR
Cowan Alfred.....2007 Pine St., Op.
Creighton, Wm. J.....258 S. 18th St., Op.
Croskey, J. W.....Med. Arts Bldg., Op.
Davis, J. L.....135 S. 18th St., ALR
Deichler, Lyn W.....1737 Chestnut St., OALR
Dintenfass, Henry.....1305 Spruce St., ALR
Embery, Frank.....4660 Frankford Ave., ALR
Ersner, M. S.....1915 Spruce St., ALR
Felt, C. L.....Chestnut & 20th Sts., ALR
Fox, L. Webster.....303 S. 17th St., Op.
Gleason, E. B.....2112 Pine St., ALR
Goddard, H. M.....1531 Spruce St., ALR
Griscom, J. Milton.....2213 Walnut St., Op.
Hawman, Erle G.....4051 N. Broad St., Op.
Heed, C. R.....1205 Spruce St., Op.
Holloway, Thos. B.....1819 Chestnut St., Op.
Husik, D. N.....1610 Spruce St., ALR
Jackson, Chevalier.....235 S. 15th St., L
Kaufman, A. S.....1923 Spruce St., ALR
Keeler, J. C.....Medical Arts Bldg., ALR
Krauss, Frederick.....1703 Chestnut St., OALR
Laessle, Henry A.....The Lenox, 13th and Spruce Sts., ALR
Lawrence, Granville A.....Medical Arts Bldg., Op.
Lewis, Fielding O.....259 S. 17th St., ALR
Loftus, John Edward.....605 Medical Arts Bldg., ALR
Lukens, Robert McD.....1923 Spruce St., ALR
Mackenzie, Alice V.....1724 Spruce St., OALR

MACKENZIE, GEORGE W.....1724 Spruce St., ALR
MILLER, E. B.....2008 Walnut St., Op.
MITCHELL, E. K.....710 W. Lehigh Ave., ALR
MOORE, WILLIAM FREDERICK.....255 S. 16th St., ALR
OLSHO, S. L.....235 S. 15th St., OALR
O'REILLY, CHARLES A.....521 Medical Arts Bldg., ALR
PETER, L. C.....N. E. Cor. Chestnut and 20th Sts., Op.
PONTIUS, P. J.....1831 Chestnut St., Op.
POSEY, WM. CAMPBELL.....2049 Chestnut St., Op.
RADCLIFFE, MCCLUNEY.....1906 Chestnut St., Op.
REESE, WARREN S.....230 S. 21st St., Op.
RIDPATH, ROBT. F.....N. E. Cor. 18th and Chestnut St., RL
ROMMEL, JOHN C.....3701 N. Broad St., Op.
SARTAIN, PAUL J.....2006 Walnut St., Op.
SCARLETT, HUNTER W.....230 S. 21st St., Op.
SCHNEIDEMAN, T. B.....2045 Chestnut St., Op.
SCHWEINITZ, GEO. E. DE.....1705 Walnut St., Op.
SENER, WALTER J.....Physicians Bldg., Op.
SHANNON, C. E. G.....135 S. 17th St., Op.
SHEMELEY, WM. G., JR.....1724 Spruce St., OALR
SKILLERN, R. H.....1811 Spruce St., R
SKILLERN, S. R., JR.....1734 Pine St., ALR
SMITH, S. MCC.....1429 Spruce St., ALR
SPAETH, EDMUND B.....N. E. Cor. 20th and Chestnut Sts., Op.
SPENCER, W. H.....1830 S. Rittenhouse Sq., ALR
STILES, CHAS. M.....1130Spruce St., Op.
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TUCKER, GABRIEL..Graduate Hosp., 19th and Lombard Sts., B
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Sturm, S. A.....7036 Jenkins Arcade Bldg., Op.
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SCHUSTER, S. A.....1st Natl. Bk. Bldg., ALR

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ISRAEL, SIDNEY.....2001 Niels Esperon Bldg., ALR
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ATKINSON, D. T.....827 Med. Arts Bldg., OALR
BURLESON, J. H.....Eye, Ear, Nose &Throat Hospital, OALR
GWINN, G. E.....704 Central Trust Bldg., OALR

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